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CARDIAC ARREST ASSOCIATED WITH SURGICAL PROCEDURES: A REVIEW OF 300 CASES*

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This paper is based on 300 cases of cardiac arrest associated with surgical operations occurring in various South African public hospitals. This report deals with fatal cases only, and they were all included in the returns of deaths associated with anaesthesia (as defined in terms of section 86 of the Medical, Dental and Pharmacy Act of 1928) submitted to the CSIR Anaesthetic Deaths Research Unit during the last 4 years (1955–1959).

Definition and Pathology

For the purpose of this article, we can define cardiac arrest as a sudden and complete cessation of cardiac function during the administration of an anaesthetic or the performance of a surgical operation, not due to irreversible shock or uncontrollable haemorrhage. By cessation of cardiac function we mean the inability of the left ventricle to pump the blood to vital organs like the heart muscle, brain, liver and kidneys.

Under this definition of cardiac arrest we include 3 specific pathological conditions, viz. (1) cardiac standstill or arrest in asystole, (2) cardiac arrest in ventricular fibrillation, and (3) feeble contractions of the myocardium not strong enough to pump the blood to the vital organs. In Table I 300 cases are classified accordingly.

TABLE I. TYPE OF ARREST (300 CASES)

	No. of Cases	% of Total
Cardiac standstill	150	50
Ventricular fibrillation	31	10
Unknown	119	40

There are 3 points which help to differentiate cardiac arrest from other causes of death associated with surgical operations. After all, every death is eventually due to a cessation of cardiac function. These points are the following: (1) The condition generally occurs suddenly and unexpectedly; (2) the actual cause of arrest is often difficult to determine; and (3) the condition is often avoidable and with prompt and adequate treatment usually reversible.

Incidence

In the absence of detailed statistics of the number of anaesthetics administered, we are not yet in a position to determine the exact incidence of this condition in South African public hospitals. We hope to be able to do this in a subsequent report. There is no doubt in our own minds, however, that this condition is on the increase, and this is in accordance with opinions expressed by overseas authors.[1-4] From our records we think 3 factors are mainly responsible for the increase during the last decade:

1. Owing to improved anaesthetic and surgical techniques more operations are being performed on bad-risk and even moribund patients, and this has led to more cases of cardiac arrest on the operating table.

2. The fact that cardiac surgery is now practised in a number of centres has also swelled the numbers; in the present series 35 cases fell in this category.

3. Modern anaesthetic techniques have been blamed by some surgeons, but we feel that lack of training and ignorance on the part of the anaesthetist responsible have more influence than the actual methods adopted.

More than one-third of our cases occurred during abdominal operations (105), with thoracic (35) and orthopaedic cases (29) taking second and third places (Table II).

TABLE II. SITE OF OPERATION[5] (300 CASES)

	No. of Cases	% of Total
ENT	7	2·3
Obstetrics	15	5·0
Genito-urinary	19	6·3
Stomach and duodenum	16	5·3
Small and large bowel	52	17·3
Other abdominal	32	10·6
Hernia (all types)	5	1·6
Thoracic	35	11·6
Endoscopy	14	4·6
Thyroid and other neck operations	5	1·6
Dental and oral	5	1·6
Orthopaedic	29	9·6
Plastic and superficial	5	1·6
Neurosurgical	15	5·0
Gynaecological	18	6·0
Other	28	9·3

Cardiac arrest is supposed to be commoner in children under 10 years and in adults over 60 years, but from the figures reflected in Table III it is obvious that no age is really immune to this catastrophe.[6]

AETIOLOGY

Hypoxia of the myocardium is usually considered as the chief cause of cardiac arrest.[7]

From this investigation it would appear, however, that excepting when the heart was pathological before operation, or in cases of asphyxia, or massive haemorrhage, *hypoxia alone* very seldom led to *a sudden cessation of cardiac function*. There were usually other factors responsible for precipitating the arrest and this will be considered in more detail presently.

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TABLE III. AGE DISTRIBUTION (300 CASES)

Decade	No. of Cases	% of Total
1	50	16·6
2	19	6·3
3	54	18·0
4	54	18·0
5	46	15·3
6	41	13·6
7	24	8·0
8	10	3·3
9	2	0·6

TABLE IV. SOME PRECIPITATING FACTORS

	No. of Cases
Pre-operative status of patients	88
Hypercapnia	70
Drug intoxication ..	61
Stimulation of peripheral autonomic nerves:	
(*a*) Vagus ..	15
(*b*) Sympathetic	3
Human factors ..	18

We also believe that when an arrest eventually occurs it is a summation of subliminal stimuli on the myocardium. These stimuli can be cumulative and additive, and for this reason short operations are probably less dangerous than prolonged procedures, especially on bad-risk patients.

In our analysis of these records we are of opinion that the factors shown in Table IV played a role in precipitating the arrest. In some cases more than one factor was considered responsible, and in others it was often difficult, and even impossible, to determine the exact cause.

1. *Pre-operative Status of Patient*

An assessment was made of the physical status of each patient before operation and all patients were classified into 4 categories A, B, C, and D as described in a previous article.[8] From Table V it will be seen that the 300 deaths include 191 poor-risk and moribund patients. Although in the moribund patient an arrest can take place at any time even without an operation or the administration of an anaesthetic, one must in some cases criticize the wisdom of operating when the prognosis is obviously hopeless from the start. We consider that in 88 cases in the list the pre-operative status of the patient was mainly responsible for the cardiac arrest. A number of these patients were suffering from thoracic diseases, and others were mostly bad-risk abdominal cases suffering from hypovolaemia and electrolyte disturbances which should have been corrected before operation.

2. *Hypercapnia*

Hypercapnia, irrespective of the cause of the accumulation of the carbon dioxide, is considered an important factor in the aetiology of cardiac arrest by most authors.[3,4,9-16] One should, however, remember that when hypercapnia occurs during the course of an anaesthetic it is usually accompanied by a certain degree of hypoxia. In this series we found 70 cases where we were of opinion that the hypercapnia (with an associated hypoxia) played a role in precipitating the arrest. The majority of these patients were either senile or suffering from chronic pulmonary or cardiac disease. We also have a clinical impression that malnourished patients who were not adequately prepared pre-operatively were more susceptible to the harmful effects of hypercapnia. Other factors responsible for this condition were inadequate ventilation of the patient during anaesthesia, especially when relaxant drugs were administered, too deep anaesthesia, and anaesthetic techniques which did not adequately remove carbon dioxide from the anaesthetic mixtures.

Hypercapnia appears to be particularly dangerous when it is accompanied by direct or reflex vagal stimulation, especially during operations in the neck and thorax. Young *et al.*[17] showed, by experiments on dogs, that the inhalation of 20% carbon dioxide and 80% oxygen until the pH of the blood is reduced may greatly augment the effect of vagal stimulation on the heart. As a contrast, it was found that a hypoxia produced by the inhalation of 10% oxygen, even when it was accompanied by hypercapnia and a low blood pH, reduced the effect of vagal stimulation on the myocardium. Stewart also found that cardiac arrest occurred very rapidly in normal dogs after hypercapnia and vagal stimulation in the thorax. In dogs suffering from chronic hypoxia before operation, the production of a hypercapnia did not augment this reflex.[15]

Another important cause of cardiac arrest associated with the accumulation of carbon dioxide is the so-called *post-hypercapniac ventricular fibrillation*. Brown and Miller have demonstrated on dogs that the danger period in association with hypercapnia follows immediately after the cessation of inhalation of high concentrations of carbon dioxide. When these dogs again inhale atmospheric air, ventricular fibrillation rapidly sets in.[13]

Sealy *et al.*[14] also investigated this condition, and came to the conclusion that hypercapnia produced in experimental animals was always accompanied by hyperkalaemia, and that the cessation of inhalation of carbon dioxide was immediately followed by a further increase of the plasma potassium. During this period ventricular fibrillation occurred very rapidly, apparently owing to the increased serum-potassium values. This may be one explanation for a sudden cardiac arrest which is sometimes reported at the end of a long and successful operation.

3. *Drug Intoxication*

An absolute or relative overdose of a drug used by the anaesthetist or surgeon during the course of an operation, or sensitivity to it, can easily precipitate a cardiac arrest on the operating table. In this category we include local analgesics, muscle relaxants, and drugs like prostigmine, adrenaline, diodine and pitocin. Polypharmacy, or the use of buckshot techniques when one drug would have served the purpose just as well, should also be mentioned in this connection.

In this series we found 61 cases where we think that the drug *per se* played a role in the aetiology of the arrest. Under this category we include 51 cases occuring during induction of anaesthesia.

As mentioned in a previous report,[8] the indiscriminate use of thiopentone (pentothal) in bad-risk patients is still mainly responsible for a sudden cardiac arrest during the stage of induction. We must again warn against the use of concentrated solutions or the rapid injection of this drug in elderly or bad-risk patients and would strongly advise the occasional anaesthetist to make use of alternate techniques when confronted with bad-risk cases. Thiopentone was the drug responsible for most of the 51 cases of arrest reported during induction.

TABLE V. PRE-OPERATIVE STATUS OF PATIENT (300 CASES)

	No. of Cases	% of Total
A (Good) ..	22	7·3
B (Fair) ..	87	29·0
C (Poor) ..	125	41·6
D (Moribund)	66	22·0

TABLE VI. PREMEDICATION

	No. of Cases	% of Total
Atropine ..	257	85·6
Scopolamine	33	11·0
Morphine ..	11	3·6
Pethidine ..	108	36·0
Other drugs	20	6·6

4. Stimulation of the Peripheral Autonomic Nerves

Cardiac arrest caused by direct or reflex stimulation of certain nerves associated with the autonomic nervous system has been reported frequently:[18]

(*a*) *Cardiac arrest from excessive stimulation of vagus nerves.* Vagal stimulation, associated with light anaesthesia and inadequate premedication, is often mentioned as a cause of cardiac arrest. We are of the opinion, however, that a fit patient, who has received an optimum dosage of atropine pre-operatively, very seldom develops a vagal inhibition during the anaesthetic, especially when muscle relaxants are used in conjunction with light anaesthesia. In this series we found 15 cases where we believe stimulation of the vagus played a part in the arrest. The majority of these cases occurred in poor-risk patients during bronchoscopies or in bad-risk cases during abdominal operations. Most of them received atropine beforehand (Table VI), but it might well be that by the time the arrest occurred the effect of the atropine had already worn off.

Sloan[19] believes that hypoxia plays an important role in the stimulation of the vagal reflex, but, from the work of Young *et al.*[7] on dogs, it would appear that carbon dioxide retention and not hypoxia is mainly responsible for augmenting this reflex during surgical operations.

(*b*) *Cardiac arrest from excessive stimulation of the sympathetic nervous system.* The usual causes of stimulation of the sympathetic nervous system during operations under general anaesthesia are oxygen lack, asphyxia, direct stimulation of splanchnic nerves, or chemical action of adrenergic substances, especially adrenaline.[7] In this respect the use of adrenaline in local analgesic mixtures in conjunction with trichlorethylene, halothane, cyclopropane or chloroform can easily cause ventricular fibrillation. We came across 3 cases where the indiscriminate use of adrenaline precipitated such an attack.

5. Human Factors

The training and judgment of the anaesthetist and surgeon and the choice of anaesthetic technique may play an important part in the final result. One cannot help feeling that some of these cases would have fared better if they had been operated on by the 'chiefs' instead of being left to junior personnel. We found 18 cases where we think that better judgment on the part of the anaesthetist might have avoided an arrest. The status of the medical personnel dealing with these 300 cases is summarized in Table VII.

In spite of all the factors mentioned above, cases still occur where it is impossible to determine the exact cause of the arrest. This may be because the data are inadequate, but, even with adequate details, we still came across 26 cases which did not fit into any of the above categories.

DIAGNOSIS AND TREATMENT

With the exception of operations in the thorax or the continuous use of an electrocardiogram, the diagnosis is usually established by the anaesthetist, and he must immediately ask the surgeon to begin the necessary treatment at once.

No time must be wasted in looking for a stethoscope in order to ascertain whether the heart is still beating. When a patient suddenly stops breathing and develops an ashen-grey colour, and the carotid artery cannot be palpated, one must assume that the patient is suffering from cardiac arrest, and cardiorespiratory resuscitative measures must be instituted immediately. Other signs are the absence of audible heart sounds, pupils which are dilating, and a blood pressure that cannot be measured. One should not, however, waste time in looking for these signs.

The only accurate method of diagnosis is an immediate thoracotomy and palpation or inspection of the heart by the surgeon. Should a diagnostic thoracotomy be done unnecessarily, free bleeding of the thoracic arteries will be found, and the incision need not be extended any further.

In most cases of cardiac arrest there is a critical time-limit of about 3 minutes in which to restore the circulation, and if one does not institute resuscitative measures *immediately*, permanent brain damage is likely to follow.

The stage at which the arrest occurred is reflected in Table VIII, from which it will be seen that approximately 1/6th of the cases occurred during induction, 2/3rds during the maintenance of anaesthesia, and 1/6th immediately after the end of the operation.

Stage at which death occurred. The majority of patients (217) were considered dead within 1 hour of the arrest (Table IX). Seventy patients succumbed within 24 hours after successful massage and another 10 patients lived for at least 24 hours.

Treatment. The detailed treatment of this acute emergency has been described elsewhere. Suffice it here is it to say that the ideal treatment consists of immediate thoracotomy by the surgeon, followed by manual massage and defibrillation where necessary, whilst the anaesthetist institutes effective measures of pulmonary ventilation. In the absence of adequate resuscitative equipment, a laparotomy and massage through the diaphragm by the surgeon, together with mouth-to-mouth breathing by the anaesthetist, is better than no treatment at all.[20] That the treatment of the majority of cases under review left much to be desired is reflected in Tables X – XIV.

TABLE VII. STATUS OF SURGEON AND ANAESTHETIST (300 CASES)

Status	*Surgeon*	*Anaesthetist*
Specialist ..	145	86
Registrar ..	55	105
G.P. ..	92	102
Intern ..	8	7

TABLE VIII. STAGE AT WHICH ARREST OCCURRED (300 CASES)

Period of Arrest	*No. of Cases*	*% of Total*
Induction ..	51	17·0
Maintenance	199	66·3
Within ½ hr. post-op. ..	50	16·6

TABLE IX. STAGE AT WHICH DEATH OCCURRED (300 CASES)

Time of Death	*No. of Cases*	*% of Total*
Under 1 hr.	217	71·6
Under 24 hrs.	70	23·6
Over 24 hrs.	10	3·3
Not stated	3	1·0

TABLE X. ROUTE OF MASSAGE (300 CASES)

	No. of Cases	*% of Total*
Abdominal ..	82	27·3
Thoracic ..	122	40·6
No massage	96	32·0

TABLE XI. COMMENCEMENT OF MASSAGE (204 CASES)

	No. of Cases	*% of Total*
Under 3 min.	87	42·2
Over 3 min.	68	33·3
Not stated	49	24·0

TABLE XII. NO MASSAGE: PRE-OPERATIVE STATUS OF PATIENT (96 CASES)

	No. of Cases	*% of Total*
A (Good) ..	5	5·2
B (Fair) ..	27	28·1
C (Poor) ..	46	47·7
D (Moribund)	18	18·7

TABLE XIII. NO MASSAGE: SUMMARY OF TREATMENT (96 CASES)

Drug	Intra-cardiac	Intra-venous	Intra-muscular	Sub-cutaneous
Adrenaline	34	5	0	1
Coramine	9	24	14	0
Noradrenaline ..	5	9	0	0
Methedrine ..	5	11	3	0
Steroids (cortisone, etc.)	0	7	2	0
Neosynephrine ..	0	1	0	0
Megamide	0	2	0	0
Picrotoxin	0	1	0	0

Massage plus injections ..	152 cases
Massage only	42 cases
Oxygen only	13 cases

TABLE XIV. MASSAGE PLUS INJECTIONS: SUMMARY OF TREATMENT (152 CASES)

Drug	Intra-cardiac	Intra-venous	Intra-muscular	Sub-cutaneous
Adrenaline	116	26	—	1
Coramine	32	47	14	—
Noradrenaline ..	25	38	—	—
Methedrine ..	6	39	3	—
Atropine	6	18	—	—
Calcium	31	4	—	—
Other drugs ..	14	59	5	—

DISCUSSION

Although the exact aetiology of this emergency is still a matter of conjecture and argument amongst surgeons and anaesthetists, there is no doubt about the treatment to be followed once an arrest has occurred. From the records we consider that some lives might have been saved had more effective measures of cardiorespiratory resuscitation been instituted immediately after the condition was noticed. Other cases, we feel, might have benefited by more efficient measures of resuscitation before operation, or by a better choice of anaesthetic technique.

As already stated, the majority of arrests (105 of the 300 fatal cases) occurred during abdominal operations, in which a number of the patients suffered from a hypovolaemia and, in our opinion, would have benefited considerably from a detailed pre-operative examination of their fluids and electrolytes followed by replacement therapy where necessary.

Except in cases of uncontrollable haemorrhage, we still believe that no operation should be attempted in the shocked patient until the systolic blood pressure has been stabilized at 100 mm. Hg and the haemoglobin raised to at least 70%.[8] In the treatment of haemorrhagic shock, one can take it for granted that when the systolic blood pressure of a normal adult has dropped below 100 mm. Hg he has lost at least 2½ pints of blood and replacement of this volume by whole blood is absolutely imperative before operation.

In considering the 51 cases of cardiac arrest during induction of anaesthesia, an overdosage of anaesthetic agent or faulty anaesthetic technique cannot be completely ruled out and the anaesthetist, therefore, may not be entirely blameless.

We are obliged again to warn against the indiscriminate use of thiopentone (pentothal) in bad-risk cases, especially by the occasional anaesthetist. When this drug is injected too rapidly, or in too large a dosage, the immediate effect may be a fatal fall in blood pressure, probably due to direct action of the drug on the myocardium.[21] If, therefore, it is used in patients suffering from hypotension or myocarditis, it is advisable to give inhalations of 100% oxygen beforehand, and inject the drug very slowly, preferably in a 1¼% solution.

Most authors are agreed that the ideal treatment of a cardiac arrest, as far as the surgeon is concerned, is immediate cardiac massage through the thoracic route.[9,20] In the 300 fatal cases under review, this was only done in 122 cases. Our main criticism, however, of the methods employed for cardiac resuscitation is that in 96 cases no attempt was made to massage the heart at all; in these cases the prognosis was hopeless right from the start. In most of them an abortive attempt was made to revive the patient by means of subcutaneous, intramuscular, intravenous or intracardiac injections. *We cannot help feeling that the injection of drugs before massage is attempted is a waste of time and should be strongly condemned in the absence of an effective circulation, as the drug cannot possibly reach the coronary arteries.*[8] Furthermore, the injection of drugs like adrenaline intracardially, especially when associated with the inhalation of agents like halothane, trichlorethylene, chloroform or cyclopropane might even be harmful and precipitate attacks of ventricular fibrillation.

We were also impressed with the variety of drugs that were being used, some of which are known not to have any effect on the myocardium. Nikethamide (coramine) may be of use as an analeptic, but it is of little value as a cardiovascular stimulant, and in cases of cardiac arrest it would be much wiser to ask the nurse for a scalpel instead of a syringe filled with the traditional ampoule of coramine.[22]

Another point worth mentioning is that of the 31 fatal cases with ventricular fibrillation, only 16 were treated with an electric defibrillator. In the remaining 15 cases there was no electric defibrillator available. As every heart may theoretically go into fibrillation after an arrest, we seriously recommend, for the attention of the authorities, the installation of defibrillation equipment in all hospitals where major abdominal surgery is being undertaken. We take it for granted that in all hospitals where thoracic surgery is being done electric defibrillators would be readily available.

In those cases in which the treatment appears to have been adequate, why did the patient not recover? We believe that the following factors were mainly responsible:

(*a*) Inexperience on the part of the staff dealing with the emergency and the lack of a proper programme of action once they were faced with an actual case. We think that all surgical and anaesthetic staff should meet at least once a year in order to become acquainted with the latest methods of cardiac resuscitation. At such a meeting a programme of action could be drawn up by the surgeons and anaesthetists and this should be displayed prominently in all localities where surgical operations are being performed.[20] If possible, cases of cardiac standstill and ventricular fibrillation together with effective methods of treatment should be demonstrated on dogs.

(*b*) In a number of cases the massage was begun too late. In order to obtain the best results the surgeon should start the actual massage within *90 seconds* of the arrest.

(*c*) Even though in some cases the massage was attempted in time, the heart might not have been pumped strongly enough to force the blood into the vital organs and maintain a satisfactory blood pressure.

(*d*) In a number of cases the patients were moribund or suffering from chronic heart disease and here the prognosis was more or less hopeless from the beginning.

(*e*) In some cases, especially in the smaller hospitals, the equipment was inadequate to deal with the acute emergency. Every hospital or nursing home, no matter how small, should have immediately available for emergency use at least a cardiac massage drum, suction apparatus, and an apparatus for applying oxygen under pressure.[20]

Finally, we should like to commend to our readers the use of hypothermia after successful massage in cases of suspected cerebral oedema or in patients with hyperpyrexia. Recently some successes were reported from this technique even in cases of arrest in excess of 5 minutes.[23,26]

REDUCTION OF MORTALITY FROM CARDIAC ARREST

As cardiac arrest appears to be on the increase, what can we do in future to reduce the alarming mortality rate which accompanies this emergency? Although the condition is not entirely preventable we feel that strict observance of the following points might assist materially in lowering the death rate:

1. *Pre-operative Period*

(*a*) A detailed history and a thorough examination of the patient before operation, especially in cases of emergency. In routine cases the patient should be examined on admission, and the blood pressure and pulse rate should again be taken immediately before induction of the anaesthetic in order to gauge the effect of fear and premedication.[24] If there is a history of the patient's having taken chlorpromazine (largactil), cortisone, promethazine (phenergan) or reserpine over a long period, appropriate pre-operative treatment should be instituted or the operation postponed.[25]

(*b*) Optimum premedication at the right time, especially with atropine, to reduce the chances of vagal inhibition.

(*c*) Correction of hypovolaemia and electrolyte imbalance before operation, especially in major abdominal and thoracic surgery.

2. *During Operation*

(*a*) The right choice of anaesthetic and the use of techniques with which the anaesthetist is thoroughly familiar.

(*b*) Careful observation of the patient with special reference to respiration, pulse rate, blood pressure and replacement of blood loss; and avoidance of hypoxia and respiratory acidosis.

3. *Immediate Post-operative Period*

(*a*) Wait until the patient is awake before injecting depressant analgesics like morphine, omnopon or pethidine.

(*b*) Unless contra-indications exist, nurse the unconscious patient on his side until he has recovered consciousness—Sims or tonsil position.

(*c*) Whenever possible, keep the patient in a recovery room until his condition is satisfactory.

SUMMARY AND CONCLUSIONS

We have given a review of the aetiology and treatment of 300 *fatal cases* of cardiac arrest associated with surgical procedures and collected from various South African public hospitals over a period of 4 years.

In a number of cases we consider the treatment was inadequate either through lack of knowledge or lack of proper resuscitative equipment.

We feel that more lives may be saved in future if every anaesthetist and every surgeon is made more acutely aware of the danger of cardiac arrest and knows exactly how to act when confronted with such an emergency. For this purpose we advocate regular meetings between surgical and anaesthetic staffs so that all members of the surgical team can receive proper instruction in the latest methods of cardiac resuscitation.

Eternal vigilance is the price of safety

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VAN DIE REDAKSIE : EDITORIAL

HODGKIN SE SIEKTE

Nog voor Virchow leukemie as kliniese entiteit benoem het, het Hodgkin reeds die siekte wat sy naam dra, beskryf in 1832. Desnieteenstaande kan 'n mens die navorsingswerk wat tot op hede op hierdie gebied gedoen is as relatief steriel beskou—uitgesonder die patologiese beskrywings en die terapeutiese resultate wat volg op röntgenbehandeling en die gebruik van stikstof-mosterd.[1]

Wat betref die etiologie van die toestand is daar groeiende bewyse dat 'n virus moontlik daarvoor verantwoordelik mag wees,[2] terwyl Rottino[1] die limfositiese en eosinofiliese hiperplasie as 'n reaktiewe verskynsel beskou wat plaasvind as gevolg van 'n tot dusver onbekende basiese verandering in die retikulo-endoteelsisteem.

Die patologiese klassifikasie is vir die klinikus van minder belang as die kliniese klassifikasie wat deur Diamond[3] en Warwick en Sellers[4] gebruik word, naamlik:

Klas I. Die siekte is beperk tot 'n enkele gebied met geen konstitusionele simptome en tekens nie.

Klas II. Twee of meer limfatiese gebiede is aangetas in 'n enkele anatomiese gebied; (*a*) sonder, en (*b*) met konstitusionele simptome en tekens.

Klas III. Verskeie anatomiese gebiede is aangetas en die konstitusionele simptome en tekens is ernstig.

Warwick en Sellers[4] stel die prognose in die groepe soos volg, bereken teen 'n oorlewingstydperk van vier jaar: Klas I 60%, klas IIa 45%, klas IIb 35%, en klas III minder as 20%. Hierdie skrywers beweer voorts dat 'n derde van die pasiënte met Hodgkin se siekte binne een jaar vanaf die diagnose sterf, terwyl dié wat die vyf-jaar en veral die tien-jaar kerf oorleef, 'n toenemende gunstige prognose het.

Die terapeutiese keuse lê tussen röntgenterapie en chemoterapie—laasgenoemde 'n ontwikkeling wat oor die afgelope vyftien jaar plaasgevind het. Hoewel foliensuur-antagoniste en kortikosteroïede beproef is, is die resultate daarmee as geheel onbevredigend. Die stikstof-mosterd groep skyn die mees doeltreffende chemoterapeutiese middels te wees. Die resultate met röntgenterapie, chemoterapie plus röntgenterapie, of chemoterapie alleen is moeilik te beoordeel. Röntgenterapie skyn nog die voorkeur te geniet vir klein groepe kliere, terwyl Warwick en Sellers[4] meen dat chemoterapie as die enigste behandeling oorweeg moet word waar die siekte te uitgebreid is vir bestraling, of waar geen verdere doel meer deur bestraling gedien kan word nie. In gevalle waar die prognose redelik goed sou wees (klas I), sou 'n mens miskien traag wees om die pasiënt onnodig aan hoë dosisse van bestraling te onderwerp, aangesien die lewensverwagting soms nog oor baie jare strek.

Chemoterapie, synde van jonger oorsprong as bestralingsterapie, hou veel verwagting vir die toekoms in. Gemak van toediening en die onnodigheid van duur en ingewikkelde toerusting by die gebruik daarvan, maak chemoterapie 'n nuttige metode van behandeling veral vir die plattelandse hospitaal en geneesheer.

Aangesien die siekte by uitstek in die twintig- tot veertigjarige ouderdomsgroep voorkom, is die gevolge op die sosiale en ekonomiese gebiede en ten opsigte van die gesinslewe veel meer ingrypend as in die geval van ander vorms van maligniteit. Om hierdie rede kan 'n mens dan ook miskien hoopvol die ontwikkeling op chemoterapeutiese gebied dophou.

1. Rottino, A. (1958): Ann. N.Y. Acad. Sci., 73, 4.
2. Bostick, W. L. (1958): *Ibid.*, 73, 307.
3. Diamond, H. D. (1958): *Ibid.*, 73, 357.
4. Warwick, O. H. en Sellers, A. H. (1959): Canad. Med. Assoc. J., 80, 423

MEDICAL INSURANCE SCHEMES

At the Meeting of Federal Council held in Pretoria on 3 - 5 March 1960, a debate lasting for more than one day of the Meeting resulted in the passing of two resolutions, each of which will have a far-reaching effect both on medical practice in this country and on the public. These resolutions read:

'That the Medical Association of South Africa agrees to cooperate with insurance organizations which provide prepaid medical care, in order to enable them to provide an adequate medical service to the public', and

'That the preferential tariff, as amended from time to time, be granted to all groups which can conform to the rules laid down for approval as a medical aid society, on the understanding that the organizations administering them will, in their turn, undertake to pay accounts of doctors direct and in full'.

For many years the Medical Association has sought to help employed persons in the middle and lower-income groups by the fostering of medical aid societies, and a considerable number of these societies exist, although they are connected mainly with larger business organizations. The new policy of the Medical Association is essentially an extension of the medical aid society principle to a greater number of the population. Numerous firms and organizations which have not had medical aid facilities in the past may now be catered for through insurance plans as long as they, as groups, conform to the rules laid down by the Association, mainly in regard to income limits. Briefly, these are as follows: The Medical Association considers that persons earning a gross income of £2,500 *per annum* or over, should be charged private practice fees, but that members of a group whose average earnings are not more than £1,100 gross *per annum* and of whose number not more than 3% earn over £1,750 gross *per annum* should be charged according to the fees laid down in the Preferential Tariff of Fees for Approved Medical Aid Societies. Approval of the society or of the group within a prepaid insurance plan by the Medical Association is necessary before the tariff becomes applicable, and the Medical Association will approve only those societies and insurance plans which are administered on a mutual non-profit basis, where any surplus funds will be used only for the extension of benefits to the members or the creation of reserve funds.

It must be emphasized that not all the insurance plans are acceptable to the Medical Association, so that insurance company plans have not been approved *in toto*. The names of those which may be approved from time to time will be published in the *Journal*, as the occasion arises.

Members of the Medical Association will be kept informed about developments through the columns of the *Journal*.

MEDIESE VERSEKERINGSKEMAS

Op 'n Vergadering van die Federale Raad wat in Pretoria gehou is op 3 - 5 Maart 1960, en na 'n debat wat meer as een hele dag van die Vergadering in beslag geneem het, is twee belangrike besluite geneem elk waarvan 'n verreikende invloed op die geneeskundige beroep in hierdie land en op die publiek sal hê. Die besluite lui soos volg:

'Dat die Mediese Vereniging van Suid-Afrika instem om hul samewerking toe te sê aan versekeringsorganisasies wat vooruit-betaalde geneeskundige dienste moontlik maak, om hulle in staat te stel om 'n toereikende geneeskundige diens aan die publiek te lewer', en

'Dat die voorkeurtarief, soos op gesette tye aangevul, aan alle groepe toegestaan word wat kan voldoen aan die reëls wat vir goedkeuring neergelê is ten opsigte van 'n mediese hulpvereniging, met dien verstande dat die organisasie wat die hulpverenigings beheer op hulle beurt sal onderneem om geneeshere se rekenings regstreeks en ten volle te vereffen'.

Die Mediese Vereniging het reeds jare lank al probeer om werknemers uit die middel- en laer-inkomstegroepe te help deur die totstandkoming van mediese hulpverenigings aan te moedig. 'n Groot aantal van dergelike hulpverenigings bestaan reeds, alhoewel hulle hoofsaaklik aan groot sakeondernemings verbonde is. Die nuwe beleid van die Mediese Vereniging is wesentlik 'n uitbreiding van die beginsel van hulpverenigings in die belang van 'n groter aantal van die bevolking. Baie firmas en organisasies wat nie in die verlede oor die fasiliteite vir mediese hulpverenigings beskik het nie, kan nou deur versekeringsplanne gedek word solank soos hulle as groepe aan die reëls voldoen wat deur die Vereniging neergelê word, hoofsaaklik ten opsigte van inkomste. Hierdie reëls is kortliks soos volg: Die Mediese Vereniging is van mening dat persone wat 'n totale inkomste van £2,500 of meer per jaar het, aan die gewone tariewe van die private praktyk onderhewig moet wees, maar dat lede van 'n groep wie se gemiddelde verdienste nie meer as £1,100 per jaar bedra nie, en van wie nie meer as 3% meer as 'n totale inkomste van £1,750 per jaar het nie, die gelde moet betaal soos bepaal deur die Voorkeurtarief vir Goedgekeurde Mediese Hulpverenigings. Goedkeuring van die vereniging of van die groep binne die raamwerk van 'n vooruit-betaalde versekeringsplan deur die Mediese Vereniging is nodig voordat die tarief toegepas kan word. Die Mediese Vereniging sal slegs die verenigings en versekeringsplanne goedkeur wat op 'n onderlinge nie-winsgewende grondslag gedryf word en waarvolgens enige oorskotte slegs vir die uitbreiding van voorregte aan die lede of vir die daarstelling van reserwefondse gebruik word.

Dit moet beklemtoon word dat al die versekeringsplanne nie vir die Mediese Vereniging aanneemlik is nie, sodat die planne van versekeringsmaatskappye nie sonder voorbehoud goedgekeur is nie. Die name van sulke planne wat op gesette tye goedgekeur word, sal in die *Tydskrif* gepubliseer word namate dit nodig mag wees.

Lede van die Mediese Vereniging sal op hoogte gehou word van ontwikkelings in hierdie verband deur mededelings in die *Tydskrif*.

INTRATHORACIC DUPLICATIONS OF THE FOREGUT

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PART I

The exact origin, formation and mode of development of duplications of the foregut are still controversial. Some explanations were advanced at a time when the nature of these anomalies was ill understood; other theories were based on the features of a single case or a particular type of abnormality and do not necessarily apply to other types. Explanations that seem to satisfy the morbid anatomy are not always acceptable embryologically. The common underlying principles are best recognized if malformations are studied in perspective, and sufficient literature has now accumulated to make this possible.

Spherical or elongated cyst-like duplications of the foregut may occur at any level from the base of the tongue to the second part of the duodenum. They are frequently classified into intramural cysts of the oesophagus, bronchogenic cysts and enteric cysts. The literature further leads one to believe that the distinction between these types according to their situation, attachments and structural characteristics is easy and accurate. My own study of the clinical, operative and histological features of duplications in 26 patients (excluding all mediastinal cysts of lymphatic and serosal origin, sequestrations, intrapulmonary cysts of bronchial origin, dermoids and hamartomas) does not altogether support these views. Of the 26, 9 were unquestionably oesophageal, 15 arose in the bronchial system, and in 2 it was impossible to ascertain the exact site of origin. A review of the literature showed that certain cases reported as teratoma or hamartoma were histologically identical with duplications described in this paper. The diagnosis in hospital records will therefore depend on the interpretation and experience of the microscopist. Thus 2 cases in the present series were initially called hamartomas.

The distribution and size of the duplications of bronchial, alimentary and undecided origin that are described in this paper, as well as the age and sex incidence of the patients, are shown in Fig. 1.

DUPLICATIONS OF BRONCHIAL ORIGIN

These are commonly called bronchogenic cysts. Laipply[47] collected 34 cases from the literature in 1945. However, these cysts are relatively common; they are mostly diagnosed on routine or mass radiography or at necropsy. They seldom produce symptoms when small, but may give rise to pressure effects when large, especially in young children, in whom they fortunately present less commonly. Usually they increase in size very slowly; several cases in this series were kept under observation for years with only slight enlargement. There was no familial incidence and no difference in the sexes. Contrary to findings recorded in the literature, the cysts occurred mainly on the right side in this series. None became infected.

Only in one case was an associated anomaly found, viz. a high dorsal scoliosis with a congenital abnormality of the spine in a boy of 14 who was X-rayed as a contact for tuberculosis. The cyst was slightly embedded in the muscle layers of the lower third of the oesophagus, but histologically it was a characteristic bronchial duplication. In another case

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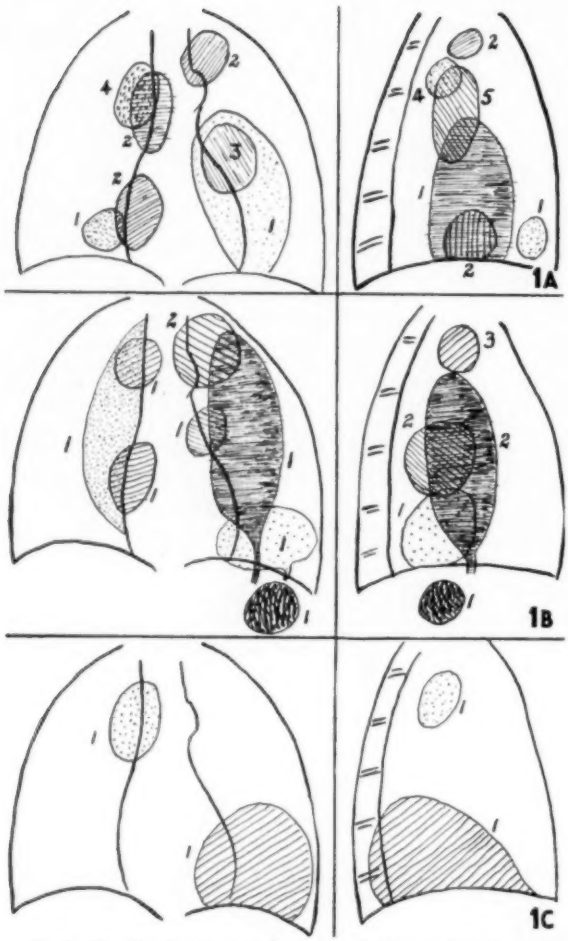

Fig. 1. The distribution and size of duplications of the foregut (diagrammatic AP and lateral drawings) in 26 patients.
(A) 15 duplications of bronchial origin in 7 female and 8 male patients at the ages of 15 months (1), 4 years (1), 5-10 years (2), 10 - 20 (2), 20 - 40 (4), 40 - 60 (4), and 65 (1).
(B) 9 duplications of alimentary origin in 4 female and 5 male patients at the ages of 5 years or under (5), 10 years (1), and between 25 and 55 (3).
(C) 2 mixed duplications in 2 males of 17 and 58 years.

the cyst in the posterior mediastinum was loosely attached to the lung, but was firmly adherent to the heads of the left 4th and 5th ribs and the vertebral column. It is possible that more of these patients had skeletal abnormalities, but in none were any special views taken of the vertebral column or spinal anomalies specially looked for. Minor degrees of rotation, especially in infants and children, make the interpretation of films often difficult, and spinal lesions which are conspicuous on some X-rays may be completely missed on others. Even tomography[79] does not always succeed in showing up the skeletal defects.

The cysts are almost always thin-walled, semi-translucent, single or multilocular, and lie characteristically in the posterior mediastinum in close relation to the trachea and main bronchi. They may be in a superficial extrapleural plane without any apparent connections and shell out with great ease, or be connected by a vestigial fibrous core or rudimentary bronchus to the tracheobronchial tree. In 2 cases the cyst wall was inseparable from the trachea in the mid-line. In one case an oval defect about 1·5 cm. long was present in 4 tracheal rings, where the cyst was adherent to the side wall of the trachea. In 2 other cases the cyst was embedded in the oesophageal wall, lying within the muscle layers of the organ. In one case (Fig. 2) the cyst was wedged in between the trachea and oesophagus so that the muscle fibres of the oesophagus had to be divided and sutured afterwards, and a fragment of the cyst wall had to be left behind on the posterior wall of the trachea, with which it was fused. In none of the cysts was there a fistulous communication with the oesophagus or tracheobronchial tree. The size of the cyst depends primarily on the degree of differentiation of its secretory epithelium and the abundance of deep glands. The contents usually consist of a milky, gelatinous, mucoid material with desquamated epithelial cells and of alkaline reaction.

The histology of these cysts is fairly uniform and the diagnosis seldom presents any difficulty (Fig. 3). The epithelium is predominantly pseudostratified ciliated columnar, but the cells may be flattened in certain areas or the cilia may be absent. It may however be stratified squamous of adult type, or resemble a more primitive stratified pattern. It may be smooth and uniform, or heaped up and irregular, or show such marked papillary formation as to suggest intestinal secretory epithelium. The epithelial lining was sometimes shed in parts or in its entirety.

The epithelium is supported by a loose connective-tissue basement membrane lying on a thin layer of irregular smooth muscle bundles of varying thickness. Fragments of hyaline cartilage were present in varying amounts, but sometimes completely absent. Pale-staining mucous glands were common within the muscle layer, but were not invariably found.

FOREGUT DUPLICATIONS OF ALIMENTARY ORIGIN

Ladd and Gross[16] instituted the collective term 'duplication' to cover the variety of interesting congenital anomalies

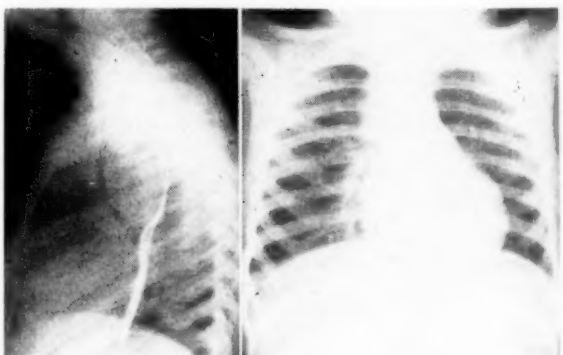

Fig. 2. The AP (B) and lateral (A) radiographs in a girl of 4 years, showing a duplication of bronchial origin in a central position between the trachea and oesophagus.

Fig. 3. The typical histological appearances of a duplication of bronchial origin, viz., pseudostratified ciliated columnar epithelium, scanty smooth-muscle fibres, pale-staining mucous glands, and cartilage.

formerly known as enteric cysts, enterocystoma, enterogenous cysts, accessory cysts, accessory thoracic stomach, archenteric cysts, etc. Although formerly regarded as curiosities of morbid anatomy, the surgical importance of these cysts is now fully recognized. Gross's criteria[31] for duplications—structures which (1) possess a coat of smooth muscle, (2) are lined by a mucous membrane similar to some part of the alimentary canal, and (3) are intimately attached to some portion of the alimentary tube—are mainly true for the intestine, but unfortunately they have been applied to the oesophagus without qualification. In contradistinction to the cysts of bronchial origin, which usually follow a benign course and are commonly diagnosed in older subjects on mass radiography, duplications of oesophageal origin frequently give rise to symptoms in infancy or childhood and may rapidly develop disastrous complications if not treated surgically.

In 1947 Lindquist *et al.*[31] collected from the literature 80 cases of intrathoracic cysts lined by alimentary-tract mucosa; 40 were in the mediastinum. All 8 cysts in the present series were mediastinal and posterior (Fig. 1). They varied from a small spherical structure 3 cm. in diameter to large multilocular and elongated sacs which almost filled the hemithorax. Some cysts were in a superficial extrapleural plane unattached to the oesophagus or other mediastinal structures (Fig. 4), but the majority were relatively thick-walled and adherent to surrounding structures. Unattached duplications were normal on barium swallow but others were so intimately connected with the oesophagus that a deformity or filling defect could be demonstrated. One cyst had a fine fibrous pedicle 3 mm. thick, which extended upwards through the thoracic inlet. Two of the cysts passed through the left hemidiaphragm and one was connected to the fundus of the stomach. None, however, communicated with a hollow viscus, although this has been described. The diagnosis is then suggested by a fluid level in the erect position or by outlining the duplication with barium.

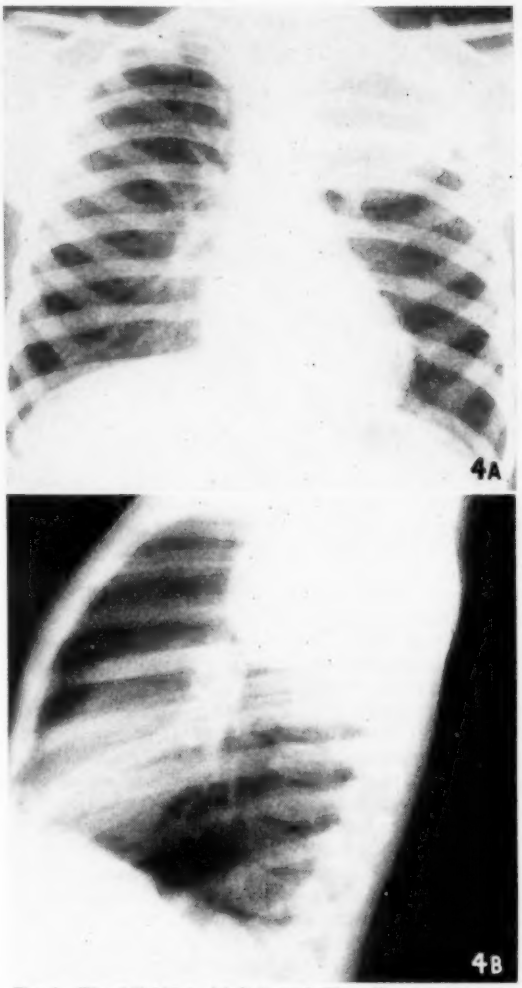

Fig. 4. The AP (A) and left lateral (B) radiographs in a boy of 6 years with a duplication of oesophageal origin.

These cysts are relatively thick-walled and may resemble the wall of the alimentary tube very closely (Fig. 5). The epithelium may be uniform throughout, appearing as a smooth lining indistinguishable from that of the oesophagus or stomach, or it may be irregular and many different types of epithelium may be present in one cyst. The squamous epithelium is often of a primitive type; the cells are large and only a few layers thick, non-ciliated and containing large pale-staining nuclei. This epithelium may become flattened and almost cuboidal, or it may change to typical pseudostratified ciliated respiratory epithelium, or become more differentiated into typical stratified squamous epithelium. In the 2 elongated cysts the squamous epithelium changed above the diaphragm to a zone of typical cardiac glands with short ducts and tortuous tubules, scanty or absent oxyntic cells, and pale-staining mucous glands. This zone quickly changed to gastric mucosa of fundic type, rich in oxyntic cells with long tubular ducts. Differentiation of squamous epithelium into gastric

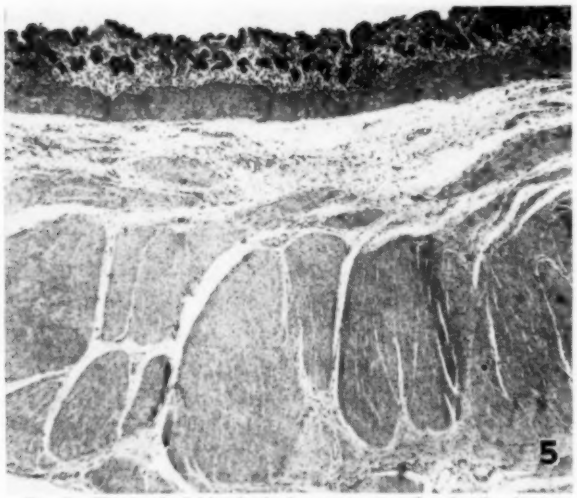

Fig. 5. The typical histological appearance of a duplication of oesophageal origin, viz., a well-defined secretory epithelium, thick muscularis mucosae, and well-developed muscle coat.

epithelium is often incomplete, so that isolated islands of gastric mucosa occur, and in 2 cases there was evidence of ulceration and chronic granulation-tissue formation around the islands of secretory epithelium. In certain areas the entire mucosa was shed. Intestinal or colonic epithelium was not found in the present series but Olenik *et al.*[59] found in a reviewed series (1946) that 12 cysts were lined with primitive oesophageal epithelium, 15 with gastric mucosa, 4 with intestinal mucosa, and 8 with a mixed epithelium representative of both the respiratory and intestinal tracts.

The muscularis mucosae (smooth muscle) is very well developed and commonly consists of 2, 3, or even 4 layers of longitudinal, circular and oblique fibres. The submucosa may be very thick and then contains numerous dilated thin-walled blood vessels and collections of lymphoid tissue, or it may be thin and ill-defined. A myenteric plexus of ganglion cells can often be differentiated, and occasionally large nerve trunks may be identified, suggestive of branches of the vagus nerves. The muscle coat is very well developed and consists of 2-5 layers of circular and longitudinal muscle fibres. The bundles are irregularly arranged, however, and lack the uniform appearance of efficiency which characterizes normal gut.

The contents varied from a mucinous opalescent fluid to a chocolate-coloured paste containing a high proportion of epithelial debris, altered blood and red cells. The pH varied from 7·8 to 3. Hydrochloric acid, enzymes and cholesterol crystals were present in varying amounts.

Associated Abnormalities

Roth,[70] in 1881, described a mediastinal enteric cyst which was firmly adherent to the vertebral column in a patient who also had an ileal duplication and a cyst of the mesentery, again illustrating the principle that congenital anomalies are often multiple. They are usually intestinal or skeletal, or both.

Skeletal abnormalities. In 1954 Fallon *et al.*[23] found 25 vertebral abnormalities (18 of which were spina bifida anterior, hemivertebra or infantile scoliosis) in a series of 61 cases which they collected from the literature, but they also pointed out that the standard chest radiograph is far from ideal for the study or even the recognition of vertebral lesions. Spinal abnormalities in the cervicodorsal area are relatively far cranial to the duplication, so that attention would only be focussed there by observers who are aware of the association. Unfortunately the cervical vertebrae are often not even on the film. The fact that skeletal abnormalities have been missed in some of the cases described in this paper, as well as in other cases of tracheo-oesophageal fistula that I have reviewed, confirms that the incidence of skeletal anomalies is higher than reported by other workers.[60,59,45]

The basic spinal lesion is incomplete anterior fusion. This may vary from minor degrees of cleft vertebra to frank anterior spina bifida, where the halves of the vertebral bodies on each side of the cleft usually diverge to form an oval passageway allowing the spinal cord to come into some form of relation with the gut. Feller *et al.*[24] in 1929 collected 28 cases of malformation which involved the central nervous system and the alimentary canal, and all of them had anterior spina bifida.[24] In my own experience this is also the commonest vertebral anomaly associated with tracheo-oesophageal fistula and the Klippel-Feil type of syndrome. Ladd *et al.*[45] mention a case of mediastinal duplication with asymmetrical thorax and short stunted neck that falls into this category. Incomplete anterior fusion may be manifested by hemivertebrae, often resulting in congenital scoliosis. It may affect a single vertebra or a series, may be on the same or opposite sides, and may co-exist with spina bifida anterior or other defects in other vertebrae. Although most of these vertebral abnormalities are found in the lower cervical and upper thoracic vertebrae, Roberts *et al.*[69] found spina bifida as high as the second cervical in one subject and a hemivertebra on the right at the level of the 7th thoracic together with a more extensive spina bifida defect of the cervical spine in another. Both these cases had elongated mediastinal duplications.

Apart from abnormal anterior fusion of the vertebral bodies there may also be, in addition or quite separately, many other skeletal anomalies. Amongst those described are cases of spina bifida posterior,[60,47] scoliosis with[58] or without[22] spina bifida anterior, and cases of fusion of the cervical vertebrae with and without kyphosis.[37,23] A case described in the present paper showed fusion of the 3rd, 4th and (partly) 5th dorsal vertebrae. Erosion and concavity of the upper dorsal vertebrae were found in one of my cases but as the cyst was large, this effect was probably the result of pressure necrosis and not primarily of congenital origin; Mixter *et al.*[55] reported a similar case in 1929. Abnormalities of the thoracic cage have been reported by Roberts *et al.*[69] and Ladd *et al.*[45] Guillery[32] described a remarkable case showing a distorted vertebra with a large mediastinal duplication on the ventral side of the vertebral column, and a smaller cyst on the dorsal side of the vertebral body; both had funnel-shaped extentions which appeared to run into the vertebral body as if to connect with one another. Other vague references to vertebral abnormalities occur.[77,1] Although there may be no radiological evidence of a spinal anomaly, dense adhesions or even fibrous stalks may connect the cysts to the vertebral bodies, posterior rib ends or paravertebral gutter;[70,5,14] several such cases occur in the present series.

Intestinal abnormalities. Intrathoracic duplications of the foregut may be associated with duplications of the stomach,[27] duodeno-jejunal flexure,[69] and ileum.[61] The present series contains 1 case with duplication of the ileum. Out of 81 cases with intrathoracic duplication reviewed in 1953, Ware *et al.*[80] found 3 with associated small-bowel duplications and 1 with a malrotation; Roberts *et al.*[69] have since reported 2 further cases with malrotation.

Mediastinal duplications may end blindly in the diaphragm[55,37,23] or in the hiatus.[62,69] They may also pierce the diaphragm[45] (there is one such case in the present series), be attached to the stomach (author's case), or open into the duodenum or jejunum.[31,25,69]

A rudimentary fissure completely dividing the apical segment from the basal segments of the left lower lobe was the only other variation from normality found in the present series.

FOREGUT DUPLICATIONS OF UNDECIDED ORIGIN

In the great majority of cases it was fairly easy to decide on the site of origin of the cyst, but in 2 cases this could not be done with certainty (Fig. 1). In one, a cyst the size of a tangerine was lying within the wall of the oesophagus in the superior mediastinum between the oesophagus and the trachea. The muscular wall of the oesophagus had to be incised and repaired after enucleation. The thick smooth muscle coat of the cyst and the mixed muscle fibres of the oesophagus merged into one. There were no bronchial glands, cartilage or muscularis mucosae, and the epithelium was cuboidal and ciliated columnar. In the other case a large multilocular cyst was found in the left lower chest, firmly adherent to the diaphragm and vertebral column and apparently arising from the lower oesophagus. The contents were chocolate-coloured and contained altered blood. The muscle wall was thin. There was no muscularis mucosae or cartilage, but numerous mucous glands had all the characteristics of bronchial glands. The epithelium was columnar, ciliated and cuboidal, and in places papillary processes closely resembled cardiac glands.

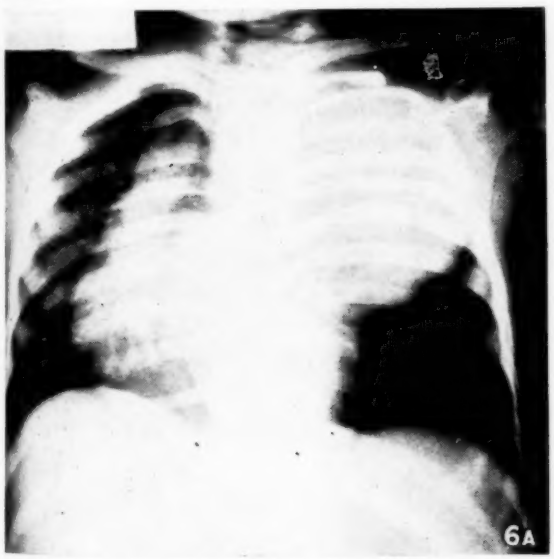

Fig. 6(A). A penetrating AP radiograph of a 2-year-old girl with a foregut duplication in the left upper chest.

ILLUSTRATIVE CASE REPORTS

Case 1

C.G., a girl of 18 months, was referred to hospital with a persistent unproductive cough which became worse after an attack of whooping cough. A chest radiograph showed a right upper mediastinal mass. Barium swallow showed the oesophagus to be pushed to the left and backwards and the trachea displaced forwards. A pre-operative diagnosis of foregut duplication was made. At operation (right thoracotomy) a smooth, thin-walled extrapleural cyst was found between the trachea and oesophagus immediately above the azygos vein. It was slightly embedded in the adventitia of the oesophagus but enucleated easily. Anteriorly the cyst wall fused with the membranous part of the trachea, and this portion had to be left behind after the trachea was inadvertently opened several times. Recovery was uneventful.

The cyst contained a gelatinous opalescent fluid; the wall was smooth and uniform. The histology was characteristic and confirmed that the duplication was of bronchial origin—pseudostratified, ciliated, columnar epithelium, smooth muscle bundles, bronchial glands, and isolated fragments of hyaline cartilage.

Case 2

P.B., a female of 47, was X-rayed for tuberculosis as a contact and a large mediastinal mass was found in the right upper chest. Tomograms showed a sharply defined edge and the diagnosis of a mediastinal cyst was made. At thoracotomy a large, thin-walled cyst was found in the posterior mediastinum, immediately extrapleural, and connected to the trachea by a rudimentary bronchus-like pedicle. The cyst was lined by a uniform smooth mucosa and contained milky mucinous material. In some areas the epithelium was completely shed; in others it was pseudostratified, cuboidal or flattened, with heaped up papillary projections. The further presence of smooth muscle bundles, cartilage, and racemose mucous glands, shows this to be a foregut cyst of bronchial origin.

Case 3

G.P., a full-term female infant of 6½ lb., was first seen at the age of 2 years after an attack of broncho-pneumonia, when a radiograph of the chest showed a left upper mediastinal mass. The Casoni, complement fixation and Mantoux tests were negative, and the blood picture showed no abnormality. Further X-rays revealed a large rounded mass in the left upper chest, encroaching on the vertebral column and eroding the posterior rib ends and the vertebral bodies (Fig. 6A). The mediastinum was pushed over to the right and the upper ribs on the left side were thin, elevated, and asymmetrical. The 3rd, 4th and (part) 5th dorsal vertebrae were fused, giving rise to a slight wedge deformity and scoliosis. A pre-operative diagnosis of foregut cyst or neurofibroma was made.

At operation a large loculated cyst was present, mainly in the superior mediastinum, which because of its size was aspirated to facilitate dissection. A milky sero-mucinous fluid (pH 8) was obtained. Firm adhesions connected the superior surface of the cyst to the extreme apex of the thoracic cavity, but it was mainly adherent to the 3rd and 4th thoracic vertebral bodies. These were concave and partly eroded, but there was no obvious extension of the cyst into the vertebral column. The aorta and oesophagus were displaced to the right. The latter was indentified by passing a bougie, but it was free from the cyst. From the inferior loculus of the cyst a prolongation passed downwards to and through the diaphragm in the neighbourhood of the hiatus, where it was ligated and divided without establishing its connections below the diaphragm.

Pathology. The duplication was 10·5 cm. long and consisted of a large spherical loculus and a smaller oval lower one, connected by a narrow isthmus. One surface was covered by pleura and the other was ragged, with numerous loose fibrous and muscular strands. On cross-section the cyst proved to consist of 3 definite and separate loculi, but intercommunicating by 2 circular smooth

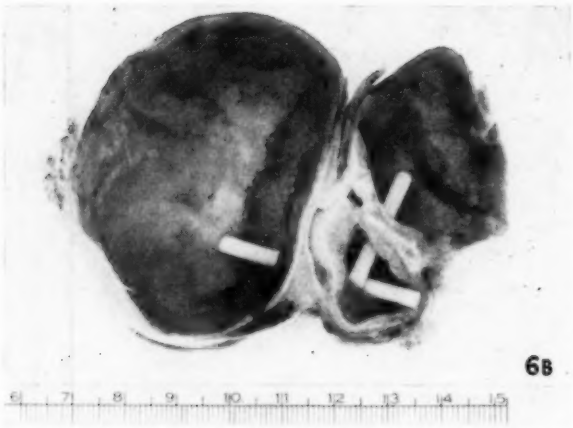

Fig. 6(B). The loculated cyst from the inside. The probes show the holes of communication.

holes 0·9 and 1·2 cm. in diameter (Fig. 6B). The muscular wall of the upper loculus was relatively thin but that of the lower 2 were thick, especially at the isthmus, where the circular muscle layer formed an almost ring-like triangular band. The lining epithelium of the upper loculus was uniform, smooth and pearly white, and vaguely resembled squamous epithelium. Near the upper hole it changed to typical velvety secretory epithelium. In the lower loculus the mucosa further changed and became circumferentially plicated, resembling the folds of Kerkring in the duodenum. Histologically, the upper part contained stratified squamous epithelium and the lower portion secretory epithelium of fundic type.

Follow-up. This child did well after the operation but in subsequent years the mother repeatedly stated that the child complained occasionally of abdominal cramps, and that she had noticed blood in the stools at times. On 2 occasions a barium meal and follow-through gave negative results. When I reviewed the child's X-ray pictures taken when she was 7 years old and

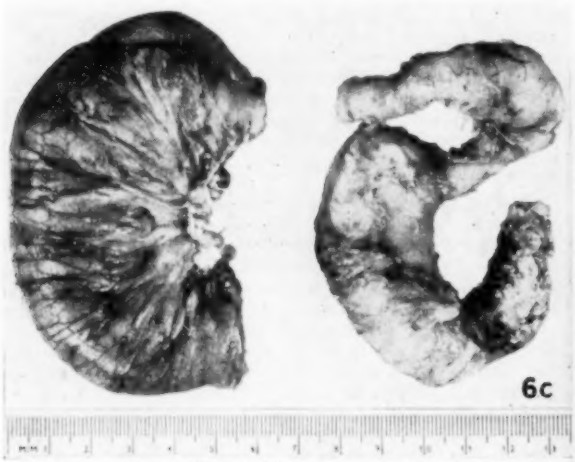

Fig. 6(C). A 10-inch duplication of the small bowel, with and without its mesentery, that was removed from the same patient 5 years after the duplication of the foregut in Fig. 6A was removed from the chest.

saw the spinal deformity together with the suggestive history, a confident diagnosis of an intestinal duplication was made and operation advised. A 10-inch duplication of the upper jejunum (Fig. 6C) was subsequently successfully removed and the child has been perfectly well since.

Case 4

R.M., born normally as a full-term infant of 8 lb., and breast fed, was admitted to hospital at the age of 3½ months. According to the mother he had stridor since birth, associated with a wheeze which she thought was due to bronchitis. Three days before admission the infant suddenly became dyspnoeic, gasped for breath and became blue. On examination he was seen to be well nourished but cyanotic; lower intercostal recession was seen on inspiration, and rhonchi were heard over both bases. A chest radiograph showed a large right mediastinal mass and immediate thoracotomy was considered advisable as an emergency measure. (A review of the films shows anterior spina bifida from C2 to T1.)

At operation the lung was free, and a large loculated cyst, not adherent to the lung or surrounding structures, was present posteriorly in the superior mediastinum. The contents from different loculi varied 'from condensed milk to brown water'. The cyst was opened in order to facilitate dissection. The mucosal lining was red and congested but no ulceration was seen. A thin fibrous pedicle extended upwards through the thoracic inlet.

Pathology. The specimen consisted of a thick-walled cyst, 10 cm. in diameter, which contained thin, brownish fluid with pH 3·9. The cyst wall, 4 mm. thick, showed thick muscular coats lined by apparently normal secretory epithelium. Histologically the epithelium was poor in oxyntic cells.

Case 5

P.L., a full-term normally-born baby girl, was first seen in hospital at the age of 3 months, with feeding trouble. Up to the age of 3 years she was never well. She then developed a left subcostal swelling, which showed up as a large, smooth filling defect of the stomach (Fig. 7A), and was admitted shortly afterwards to another hospital, where a left subphrenic abscess which had ruptured into the left pleural cavity was diagnosed and drained through the chest and abdomen. The post-operative recovery was stormy, but she was much improved for the next 3 years except for periodic vomiting and pain in the left subcostal area. At the age of 6 she again became ill, developing a high fever with signs of left basal pleurisy and pericarditis. The rapidly increasing pericardial effusion was aspirated and brownish offensive fluid obtained, which cultured *B. coli* organisms. Despite antibiotics and aspirations the child died shortly after transfer to the thoracic surgical unit.

Post mortem examination revealed an oval duplication (6 × 4 cm.) on the greater curve of the stomach fundus. A 2-cm.-wide chronic ulcer had perforated into the left posterior subphrenic space, through the diaphragm into the pericardium, resulting in a diffuse

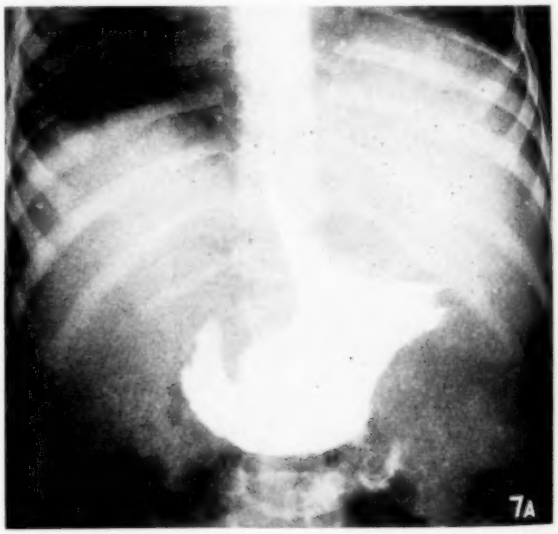

Fig. 7(A). On barium-meal examination a large, smooth, round filling defect of the upper stomach was seen in this 6-year-old girl.

Fig. 7(B). Voluminous mucosal folds are present on the inside of the stomach, where the duplication was attached to the greater curvature.

fibrinous pericarditis. The cyst was covered by peritoneum on the outside and was entirely free from the stomach except at one point, where it was connected by a flimsy fibrous pedicle. On the inside of the stomach at this point voluminous irregular redundant mucosal folds were conspicuous (Fig. 7B). The muscular wall of the cyst was much thicker than that of the stomach. Histologically the mucous membrane was typically of fundic type. The ulcer had all the characteristics of a chronic peptic ulcer. There was no other abnormality and no evidence of a spinal lesion.

Case 6

D.C., a normal healthy boy of 6 years, was X-rayed for tuberculosis as a contact and a mass was found in the left upper chest. Routine investigations threw no light on its nature and a preoperative diagnosis of neurofibroma or mediastinal cyst was made. (Review of the X-ray showed anterior spina bifida in the 4th and 5th cervical vertebra.)

At operation a large spherical cyst was found in the superior mediastinum, close to the oesophagus, but completely unattached to it, the trachea or the spinal column. It enucleated easily, leaving a clean surface.

Pathology. The cyst was thin-walled (about 1 mm. thick in most places) and measured 8 × 5 cm. It contained a brownish fluid, which proved to be partly altered blood. At one point there was a slight thickening in the wall, which presented externally as a boss measuring 1·5 × 1·2 cm. and about 2 mm. above the general surface. Internally it was pinkish, and centrally umbilicated, with the central fossa about 6 mm. in diameter and 2 mm. deep and the plaque raised about 1 cm. above the general internal surface. This island contained velvety mucosa similar to that of the body of the stomach, and rich in oxyntic cells. At the edge of this island were the appearances of a chronic gastric ulcer, with well-marked fusion of the muscularis mucosae and the outer muscle layers. It was this surrounding fibrosis which buckled the patch of the stomach to give the internal concave surface. Auerbach's plexus was well developed. A few centimetres from the boss there was a large nerve, containing ganglion cells; also a small artery, a few ganglia and a lymph node. The epithelial lining of the rest of the cyst was mostly shed; a few islands of stratified squamous epithelium remained. The muscle coat was well developed in 3 layers at the island of gastric mucosa, but the rest of the cyst was rather fibrous, the muscle layers being irregular, thin and undeveloped.

Case 7

E.M., a man of 51, was admitted to hospital because of recent haemoptysis and pain in the left lower chest. Chest radiographs showed a rounded mass posteriorly in the left lower chest.

At operation, a thick-walled cyst was present in the left paravertebral gutter, firmly adherent to the vertebral column, but only loosely attached to the lung. A prolongation extended downwards through the diaphragm, which was incised and found to be attached to the fundus of the stomach. Although the muscular coats fused, the mucosal linings were separate and no communication was demonstrable between the cyst and the stomach. The prolongation was excised from the stomach and the entire cyst removed.

Pathology. The specimen showed a spherical cyst 10 cm. in diameter with a small prolongation on the one side. It was partly covered by pleura. On the inner and posterior aspect there were 2 bosses, 4·5 × 0·4 cm. and 1·4 × 1·5 cm. respectively, separated by a gap of 0·7 cm., and composed of mucus-containing ectopic lung tissue. These 'ectopic lungs' were supplied by a large, thick-walled systemic artery of 2 mm. diameter and two smaller vessels of 1 mm. each. Non-pigmented 'hilar' glands were present where the vessels entered the bosses. The bronchus contained cartilage and racemose mucous glands of bronchial type. There was no actual communication between the cyst and the ectopic masses of lung.

The wall of the cyst was very thick (4 - 5 mm.). The mucosa was smooth but changed in appearance at about the level of the diaphragm. There was no evidence of ulceration. Histologically the cyst was lined by ciliated columnar epithelium. At the diaphragmatic level the cilia were lost, and the mucosa changed to a single layer of columnar cells, which then changed to a secretory epithelium typical of the zone of cardiac glands, and finally to typical fundic mucosa, rich in oxyntic cells. The muscularis mucosae consisted of 3 - 4 layers, while the outer muscle coat contained up to 5 separate layers.

Case 8

S.W., a normally-born full-term baby girl was admitted at the age of 14 months with a history of difficulty in breathing since the age of 4 months. A chest radiograph showed a mass in the right upper mediastinum and barium swallow indicated a smooth uniform filling defect on the right side immediately above the tracheal bifurcation. Below this level opposite the mass the oesophagus was kinked and pulled sharply to the right. A diagnosis of oesophageal duplication was made.

At operation a small cyst was found within the muscular wall of the oesophagus immediately above the azygos vein. It had no connection with the trachea or spinal column, but the muscle layers of the oesophagus had to be incised in order to enucleate the cyst.

Pathology. A spherical, smooth, thin-walled cyst of 3·5 cm. diameter was found containing an opalescent thin mucinous fluid. The mucosa was pale and uniform. Histologically the wall consisted of a small muscle layer of striated fibres and a thin muscularis mucosae. The epithelium was ciliated stratified columnar. Cartilage and bronchial glands were absent.

(To be continued)

SOUTH AFRICAN PAEDIATRIC ASSOCIATION (M.A.S.A.), NATAL SUB-GROUP

A Sub-Group of this Association has been formed in Natal and the following office bearers elected: Chairman, Dr. H. L. Wallace; Vice-Chairman, Dr. F. Walt; Hon. Secretary, Dr. N. M. Mann, Department of Medicine, University of Natal; Committee, Drs. P. Klenerman and F. Friedlander. All medical practitioners in Natal who are interested in paediatrics are welcome to join this Sub-Group as Associate Members and should communicate with the Secretary in regard to this matter.

THE PSYCHIATRIC APPROACH TO TUBERCULOSIS*

B. CROWHURST ARCHER, M.D., *Durban*

The behaviour of a patient and his attitude towards an illness will depend upon what he thinks about his illness, what he considers to be its cause, and how he believes others regard him as a diseased person.

Coleman[1] has pointed out that what happens to the personality in an illness depends on a number of factors, including age and sex; the parent-child relationship (in children); the nature of the symptoms—the way they develop and the after-effects; the course of the illness and the extent of its threat to life and physical integrity; and, finally, the folklore associated with the particular illness which, for each individual, becomes translated into a kind of personal mythology.

The psychiatrist, as a physician, is concerned not only with the function of the individual organs of the human body, but also with the health of the human being as one integrated and inseparable whole. Moreover, he is interested in the social forces which facilitate or retard emotional adjustment in the individual and in the group.

This multi-disciplined approach to the problem of illness attempts to coordinate thinking in the various biological sciences with the relevant psychological and social data, in order to explore the complexity of the disease-reaction, rather than reduce it to the conceptual framework of any particular discipline. I believe that this approach merits serious consideration in all illness; particularly in a chronic illness like tuberculosis which has proved resistant to approaches based on the tenets of a single discipline.

EMOTIONAL FACTORS

Holmes[2] said that cultural conflict contributed significantly to the natural history of tuberculosis. He contended that the patient's emotional adjustment almost invariably broke down 2 years before the onset or relapse of the disease. It was the increased stress of life upon individuals, who were no longer able to resolve their emotional problems or achieve satisfaction in life, that precipitated the development of tuberculosis.

Day[3] pointed out that tuberculosis differed from the ordinary psychosomatic diseases such as peptic ulcer and hypertension, where, presumably, an emotional habit caused symbolic functional changes which, in turn, if they persisted, caused organic physical changes. Not everyone, he said, can develop tuberculosis 'to order'. Tuberculosis was found in persons of all grades of intelligence in whom it differed again from such psychosomatic disorders, as migraine and asthma; to 'qualify' for these diseases 'it is almost essential to be up to school certificate standard'.

Coleman[4], on the other hand, said that no particular type of personality was particularly prone to tuberculosis, nor was it possible to show unequivocally that emotional factors played an important role in its pathogenesis, although there were instances in which the illness seemed to have arisen in close association with states of tension and stress. It is interesting to record that a higher percentage of obsessive compulsive personalities are found among tuberculous patients than in the population as a whole.

There are, however, typical emotional responses to the diagnosis and treatment of tuberculosis which are of importance to both the clinician and the hospital administrator. For example, when a patient first learns that he is suffering from tuberculosis, he often refuses to accept the fact of illness or, if he does, only accepts it intellectually and does not prepare himself for a new mode of life or an altered daily routine. The common emotional reactions to this insidious disease is a mild form of anxiety and depression and, as a defence, the patient often develops an attitude of defiance and ultra-cheerfulness, or a kind of repressed hostility which expresses itself in a general 'touchiness' towards the nursing staff and his associates, not unlike the reaction commonly seen in those suffering from essential hypertension. Apathy is rare and occurs in early and advanced stages of the disease and represents, if not a toxic origin, a withdrawal from an otherwise unbearable situation. Finally, since enforced rest is such an essential principle of the treatment of tuberculosis, protracted hospitalization itself tends to create a disabling neurotic condition.

THE HOSPITAL

The diagnosis of tuberculosis is always a shock to the patient. However, if the family doctor handles the situation satisfactorily, the patient will enter hospital with some anxiety, but also with the hope of recovery. It is therefore essential that the physician should understand the personality of the patient—his capacity for personal adjustment and his various emotional stresses—if he is to help him settle down in the hospital environment, maintain his hope of recovery, and prevent his irregular discharge.

The physician should appreciate that if he does little to ameliorate his patient's attitude of rebellion, suspicion and quarrelsomeness, then hospital life will become miserable for the patient as well as for those who have to deal with him; irregular discharges will occur and future rehabilitation will become impossible. If, however, the physician is sensitive to the personal problems of his patient, he will find that the common emotions of fear, inferiority, disgust, guilt, and hostility are capable of considerable modification.

Many nurses are genuinely interested in their patients as human beings and are puzzled by the behaviour problems they present. However, partly because of thoughtlessness and partly because of lack of training in matters of elementary psychology, nurses leave much to be desired in the psychological handling of their patients. Nursing tuberculous patients is not always very interesting; it could be made much more interesting for nurses if the human factor were introduced into their training.

Tuberculous patients rarely leave hospital against the advice of their doctors on the grounds that they are dissatisfied with the medical treatment. Their reasons for leaving can generally be attributed to emotional problems. It should be remembered, however, that the patients who remain in hospital still have to handle the same kinds of feelings which motivated the others to leave. To meet this difficulty, Wilmer,[5] a psychiatrist, who had himself made a satisfactory recovery from pulmonary tuberculosis, demonstrated the value of

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, September-October 1959.

group therapy. He showed that realistic acceptance of membership of a small group permitted easier acceptance of membership of the larger hospital group, as well as a more mature acceptance of the disease and its meaning by the patients' family and friends. Moreover, this approach facilitated the rehabilitation of patients after their discharge from hospital.

REHABILITATION

Rehabilitation is one of the most pressing challenges o modern medicine. Rehabilitation requires team work—not the team work of a conference, but team work in action. It involves physicians and relatives, labour unions and employers, public health nurses, and social agencies. It requires more than goodwill and a promise to cooperate. Rehabilitation begins, as Coleman[6] said, with orientating the patient to his illness. It includes realistic evaluation of the organic and emotional factors in the patient, and the social, vocational and psychological factors in the community to which he is returning. It includes a concrete plan for out-patient treatment, help in finding employment, help in overcoming prejudice, facilities for maintaining physical and mental health, vocational re-training—if indicated—and some technique for gradually 'hardening' the patient psychologically as well as physically to the non-protective climate of the outside world. To permit the patient to plunge into a rushed life which will re-activate the disease, is folly, and to restrict him so narrowly that he is hardly able to participate in the events of the world, is also folly. To help him steer a middle course is the real solution.

The social worker can make a unique contribution to the rehabilitation of the patient. In addition to her usual functions, she has the special task of helping to interpret the nature of this infectious disease to the public and the family. She also has to mobilize the community resources in vocational placement and in public health. In addition, since tuberculosis has a social as well as a pulmonary pathology, the social worker should also be helpful in assisting in the reduction of premature discharges from hospital, many of which are due to social, environmental and family factors.

Well-organized rehabilitation within the community is, in fact, an efficient method of group therapy on a large scale. The invalid recognizes that he is emotionally, socially and economically no longer an outcast, but that he 'belongs' somewhere, and that something constructive is being done for him.

Recent advances in the treatment of tuberculosis in well-institutionalized European countries, have given rise to new hope that the 'scourge of the White man' will be eliminated in the foreseeable future. The average duration of stay in hospital has been reduced by means of modern drugs to about 3 months, and the emphasis today is on rehabilitation.

THE AFRICAN

What is the position regarding tuberculosis in the African? In Natal, for example, 12·37 per 1,000 of the African population contract the disease. In the African the insidious onset of tuberculosis, which is so common in the European, is the exception; for him it is often an acute fulminating disease with a strong tendency to miliary dissemination.

What is the psychological reaction of the African to a diagnosis of pulmonary tuberculosis as compared with the average European? He often cares very little and would abscond from the ordinary hospital the moment he felt better. The problem of treating these patients successfully seems almost impossible, especially in view of the fact that with the aid of modern drugs the temperature can usually be reduced to normal within 3 weeks, and the drugs themselves often induce a mild state of euphoria.

It should be remembered, however, that the cultural background of the African is a patriarchal society composed of large family groups governed by primitive taboos. Infringement of these rules implies antisocial motives, and in this setting, even solitary and outstanding people are suspect. Logic, speculation, and the search for causes are supplemented by magic and animism and, when these fail, only the witch doctor and the ancestral spirits can help. The spirits, moreover, can be approached only by the group and not by an individual.

Gallais and Planques,[7] observing the African from a European frame of reference, state that the most striking traits of the African are the recognition of the importance of physical needs (nutrition and sexuality), and a liveliness of the emotions counterbalanced by their short duration. The African lives essentially in the present (in a sense, like a child) and his behaviour is largely motivated by influences and impulses of the moment.

The African, with this cultural background, has now been subjected to the impact of Western civilization. The indentured African has left his kraal and has embarked on a venture into, what appears to him, a rather gay, rather irresponsible meaningless world, with the restraints of his normal environment in abeyance. Finally, he exists on a diet lacking in a variety of constituents necessary to maintain his physical and mental health, and he faces life with lowered resistance to disease and with general impairment of efficiency.

Pulmonary tuberculosis in the African is often a family affair as well as a family epidemic and everything possible should be done to meet this situation if the patient is to continue his treatment in hospital. To this end the wards of the admission blocks of some hospitals where Africans are treated are only separated by walls 3 feet in height so as to preserve the sense of community life. As soon as the patient's temperature is normal, which is usually about 3 weeks after admission, he is transferred to attractively equipped open wards surrounded by pleasant lawns, and visitors are encouraged. Occupational therapy, and not group therapy—which is quite unnecessary—is prescribed, and an incentive is provided by a definite system of remuneration.

Dormer *et al.*[8] have recently reported that in King George V Hospital in Natal, with over 1,000 beds for African patients with pulmonary tuberculosis, about 20 babies are born each year. Until 1955 these babies were immediately separated from their mothers and sent to other institutions or foster parents or relatives. However, when the feeding is not carried out by the mother of the child, a very high mortality results. While it is always desirable, where possible, to keep a mother and child together, in a backward community it is life-saving. The mothers who have their children with them are contented and the babies are beautiful, thriving children.

From their experience over a period of 3 years, Dormer *et al.*[8] feel that isoniazid, given prophylactically, can completely protect a child from infection with tubercle bacilli. A child treated prophylactically does not develop any immunity and, as soon as the administration of the drug is stopped, the

child will, if exposed, become re-infected. These authors stated that isoniazid was given prophylactically to 98 newborn infants so that they might remain with their mothers in a tuberculosis ward. Given regularly, the prophylactic use of isoniazid has completely prevented tuberculous infection occurring in these children. The mothers have been allowed to breast-feed and fondle their children as much as they like, without adverse effects on mother or child.

CONCLUSION

Specialization has brought many triumphs to medicine. It has also brought one evil—the curious departmentalization of the human body. The victim is often the patient who has been so unfortunate as to acquire two diseases which fall under the jurisdiction of different specialists or, what may be even worse, two government departments.

It would be a catastrophe if this anomalous situation were to create the feeling in the minds of general practitioners that tuberculosis is the responsibility of others.

The practitioner deals with sick people, and tuberculosis is more common among those who feel ill than among those who feel well. The practitioner is the man who first sees the sick person and who knows his environment; therefore, the general practitioner, who is constantly aware of the threat of tuberculosis, remains the mainstay in the fight against this disease.

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ELEKTROKARDIOGRAFIESE STUDIES VII

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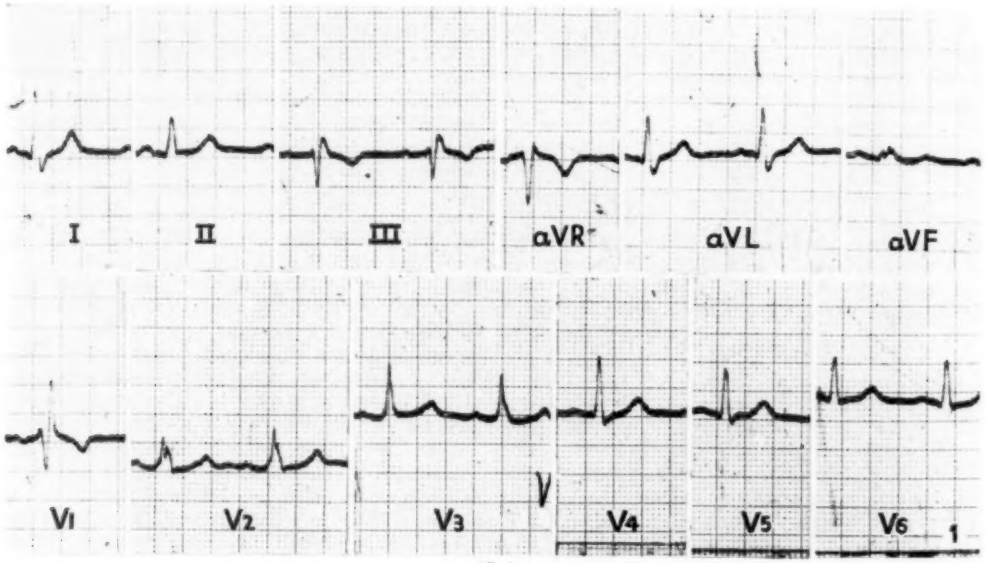
Geval 7. Paroksismale Hartblok en Ventrikulêre Stilstand met latere ontwikkeling van Volledige Hartblok

'n Blanke manlike pasiënt, 60 jaar oud, is toegelaat tot die urologiese afdeling op 13 Augustus 1959 om 'n prostatektomie te ondergaan. Benewens sy urologiese klagtes en bevindings het hy ook gemeld dat hy sedert Januarie 1956 toevalle van floute kry. Op navraag blyk dit dat dié toevalle geen spesiale eienskappe het nie, behalwe dat hulle bestaan uit kortstondige, skielike episodes van bewussynsverlies. Daar is geen epileptiese eienskappe aan verbonde nie. Die toevalle van sinkopie het voorgekom onder omstandighede van rus of beweging en was so kortstondig van duur dat hulle geen noemenswaardige ongemak veroorsaak het nie.

Algemene fisiese ondersoek het geen positiewe afwykings van betekenis gelewer nie. Polsspoed 86 per minuut. Polse was almal goed tasbaar en daar was geen duidelike bevindings wat gedui het op arteriosklerose nie. Bloeddruk 150/85 mm. Hg.

Ondersoek van die hart en hartvatstelsel het veral normaal voorgekom.

Röntgenfoto's van die borskas. Die aorta het effens verwyd voorgekom. Verder het die hart en longe 'n normale röntgenologiese beeld getoon.



Afb. 1

Die pasiënt het sy operasie ondergaan sonder enige komplikasies. Tien dae na die operasie het hy egter skielik 'n toeval van bewussynsverlies gehad terwyl hy in die bed gelê het. Hy het blykbaar geskok voorgekom. Hy het 'n polsspoed van 20 tot 30 per minuut gehad toe sy toestand onder die aandag van die verpleegsters gekom het. Daar was geen bloeddrukbepaling nie en hy het na ongeveer 20 minute weer normaal gelyk.

Tydens die mediese ondersoek wat hierop gevolg het, is daar weer geen afwykings gevind nie.

ELEKTROKARDIOGRAM

Afb. 1: Daar was 'n sinusritme teen 70 per minuut. PR-tyd was 0·18 sek. QRS toon 'n patroon van regter-bondeltakblok. ST-segmente en T-uitwykings kom normaal voor. Behalwe vir die beeld van regter-bondeltakblok is daar geen ander geleidingstoornis nie. Daar is geen tekens van miokardiale hipertrofie of isgemie nie. Daar is geen aanduiding van onlangse of ou miokardiale infarksie nie.

Na hierdie episode het die pasiënt weer twee geringe toevalle van sinkopie gehad. Gedurende November 1956 het hy 'n derde toeval gehad en daar kon klinies vasgestel word dat 'n periode van asistolie met terugkeer na normale sinusritme plaasgevind het. Hy is daarna met gereelde tussenposes gesien en is behandel met efidrien, 15 mg. 4-uurliks. Sy polsspoed het om en by 80 per minuut gebly by elke hospitaalbesoek.

Gedurende Mei 1957 is dit opgemerk dat sy polsspoed 32 per minuut was en die pasiënt het toe beweer dat hy reeds 'n paar maande lank geen toevalle van sinkopie gehad het nie. Sedertdien is hy nog gereeld onder toesig. Sy polsspoed bly stadig, ongeveer 30 per minuut, en hy het geen verdere episodes van bewussynsverlies gehad nie. Sy bloeddruk is ook gehandhaaf by 150/100 mm. Hg. en daar is geen ander kardiovaskulêre afwykings nie.

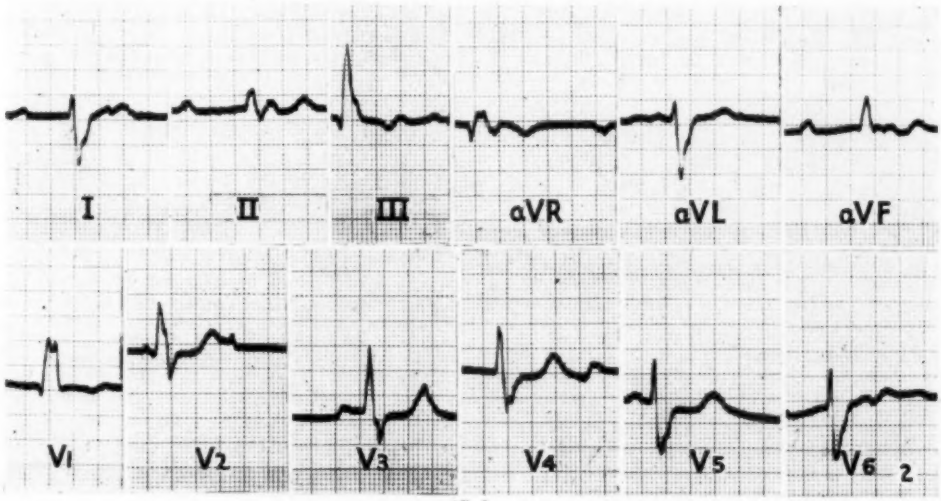
Die elektrokardiografiese beeld het vanaf Mei 1957 onveranderd gebly (Afb. 2) en is dié van volledige hartblok met 'n meer uitgesproke 'n beeld van regter-bondeltakblok.

BESPREKING

Episodes van Adams-Stokes toevalle het by hierdie pasiënt voorgekom terwyl hy tussen die aanvalle normale sinusritme gehad het. Maande later eers het daar permanente volledige hartblok ontstaan. Hierdie verloop van sake word ook deur ander vermeld,[1] maar is 'n seldsame verskynsel en is ook bekend as paroksismale ventrikulêre stilstand.[2] 'n Kliniese diagnose is uiters moeilik onder hierdie omstandighede en die toestand mag verkeerdelik gediagnoseer word as 'n geval van histerie, epilepsie of as vaso-vagale toevalle. Geringe afwykings in die elektrokardiogram mag die vermoede verwek van die werklike oorsaak van die toevalle, soos die teenwoordigheid van 'n vol of verlengde PR-tyd of volledige of onvolledige bondeltakblok, hoewel hierdie bevindings op sigself nie enige direkte betekenis mag inhou nie. Alle pogings moet dan aangewend word om een van hierdie toevalle persoonlik te kan waarneem.

Die meganisme van die sinkopie-toeval is die onvermoë van die ventrikel om saam te trek gedurende die verloop van 'n volledige hartblok, of wanneer daar 'n vertraging is in die ontstaan van idio-ventrikulêre ritme gedurende die oorgangstydperk van 'n normale sinusritme tot 'n gedeeltelike of volledige hartblok.[2] Skynbaar is laasgenoemde die meganisme soos dit by hierdie pasiënt voorgekom het. Soortgelyke Adams-Stokes toevalle mag egter ook plaasvind gedurende periodes van ventrikulêre tagikardie of fibrillasie, met of sonder geassosieerde volledige hartblok.[3]

Sinkopie-aanvalle mag ook voorkom as gevolg van hartstilstand by pasiënte met andersins 'n normale harttoestand, as gevolg van vagale reflekse, veral vanaf 'n sensitiewe karotis sinus. By hierdie pasiënt het die teenwoordigheid van 'n beeld van bondeltakblok waarskynlik die vermoede verwek van 'n moontlike hartstilstand as oorsaak vir die sinkopietoevalle, hoewel die beeld van bondeltakblok wat volledige atrio-ventrikulêre blok mag voorafgaan, gewoonlik meer 'n linker-bondeltakblok is.[4] Die latere ontwikkeling van 'n volledige hartblok het die onderliggende geleidingsfout verder bevestig. Dit is betekenisvol dat daar geen verdere sinkopietoevalle was vandat die idioventrikulêre ritme stabiliteit bereik het nie.



Afb. 2

SUMMARY

A patient, aged 60 years, complained of mild syncopal attacks for some months. It subsequently transpired that these were Adams-Stokes seizures as a result of asystole which occurred during the course of normal sinus rhythm. This condition has been termed paroxysmal heart block and ventricular standstill.

The only possible indication of the underlying disturbance of conduction was the presence of right bundle-branch block.

The patient subsequently developed complete heart block and with an established idioventricular rhythm had no further episodes of syncope.

VERWYSINGS
1. Penton, G. B., Miller, H. en Levine, S. A. (1956): Circulation, 13, 801.
2. Lawrence, J. S. en Forbes, G. W. (1944): Brit. Heart J., 6, 53.
3. Parkinson, J., Papp, C. en Evans, W. (1941): *Ibid.*, 3, 171.
4. Wright, J. C. (1955): Cardiologia (Basel), 27, 1.

SOUTH AFRICAN PAEDIATRIC ASSOCIATION (M.A.S.A.)

FOURTH SOUTH AFRICAN PAEDIATRIC CONGRESS — VISIT OF PROF. G. FANCONI

The 4th South African Paediatric Congress will be held in Cape Town on 4-6 April 1960 at the Red Cross War Memorial Children's Hospital. Interested doctors will be welcome as guests at the scientific proceedings, a programme of which will be available at the end of March, on application to the Hon. Secretary of the South African Paediatric Association, c/o Department of Child Health, University of Cape Town.

Prof. Guido Fanconi, Ordinary Professor of Paediatrics at the University of Zurich, is coming to South Africa at the invitation of the Paediatric Association, to attend this Congress, where he will present a paper on 'Some aspects of the physiology and pathology of calcium and phosphorous metabolism'.

While in South Africa, Professor Fanconi will address various meetings of members of the medical profession, details of which are given below. It is suggested that interested doctors enquire at their local Branch for details of the venues of these lectures.

Johannesburg, Tuesday 29 *March*

'Prenatal pathology: Some aspects of hereditary diseases, chromosomal aberrations and post-conceptional pathology' at 8.15 p.m. in the Harveian Lecture Theatre, Medical School, University of the Witwatersrand. In conjunction with the Southern Transvaal Branch (M.A.S.A.) and the Medical Graduates Association of the University of the Witwatersrand.

Pretoria, Wednesday 30 *March*

'Electrolyte and water therapy' at 8.15 p.m. In conjunction with the Northern Transvaal Branch (M.A.S.A.).

Bloemfontein, Friday 1 *April*

'Prenatal pathology: Some aspects of hereditary diseases, chromosomal aberrations and post-conceptional pathology' at 8.15 p.m. In conjunction with the Orange Free State and Basutoland Branch (M.A.S.A.).

Cape Town, Monday 4 *April*

'Prenatal pathology: Some aspects of hereditary diseases, chromosomal aberrations and post-conceptional pathology' at 8.15 p.m. in the Physiology Lecture Theatre, Medical School, Observatory, Cape. In conjunction with the Cape Western Branch (M.A.S.A.).

Port Elizabeth, Friday 8 *April*

'Electrolyte and water therapy' at 8.15 p.m. In conjunction with the Cape Midlands Branch (M.A.S.A.).

Durban, Monday 11 *April*

'Problems of nephrosis' at 8.15 p.m. In conjunction with the Natal Coastal Branch (M.A.S.A.).

IN DIE VERBYGAAN : PASSING EVENTS

Pensioene vir Persone wat vir Hulself Werk. Lede van die Mediese Vereniging sal belangstel in die verwysing in sy Begrotingsrede deur die Minister van Finansies na 'n vergunning van £300 per jaar, wat beskikbaar is vir pensioendoeleindes in die gevalle van persone wat vir hulself werk. Baie pensioenskemas sal waarskynlik in die onmiddellike toekoms aan praktisyns voorgehou word, maar lede word versoek om dit in gedagte te hou dat die Vereniging besig is om die hele saak van 'n geskikte pensioenskema wat van toepassing op private praktisyns is, te ondersoek. 'n Aankondiging sal hopelik mettertyd in die *Tydskrif* gemaak word.

* * *

Found. A cigarette lighter was found in the hall after the recent Meeting of Federal Council in Pretoria. Will the owner please apply to the Secretary, P.O. Box 643, Cape Town.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.) Joint Lectures. The next lecture in this series will be held on Wendesday 23 March at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Prof. I. Boerema, of Amsterdam, will lecture. All members of the Medical Association are welcome.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 28 March at 5.10 p.m. in the Institute Lecture Theatre. Dr. B. Wolstenholme will speak on 'The bilharzia complement fixation test'.

* * *

East Rand Branch (M.A.S.A.), Jooste Cup Golf Competition. The annual golf competition 'The Jooste Cup', run by this Branch of the Association and open to all members, is being held this year at the Springs Country Club on 10 April. Entries may be submitted to the Convener, Dr. W. Sacks, at P.O. Box 41, Springs, or at telephone 56-5371 (office) and 56-1138 (residence).

Dr. Louis A. du Plessis, torakschirurg, van Johannesburg, het na Londen toe vertrek waar hy die British Council se kursus oor hartchirurgie sal bywoon vanaf 20 Maart tot 2 April. Dr. du Plessis sal ook verskeie ander sentrums in Engeland en Frankryk besoek waar hartchirurgie gedoen word. Hy sal op 18 April weer in Suid-Afrika terug wees.

Mr. Louis A. du Plessis, thoracic surgeon, of Johannesburg, has left for London where he will attend The British Council's Cardiac Surgery Course from 20 March to 2 April. Mr. du Plessis will also visit other centres in England and France where cardiac surgery is performed. He will return to South Africa on 18 April.

* * *

Society for Clinical and Experimental Hypnosis of South Africa (Northern Section). A symposium on hypnosis will be held at Medical House, Esselen Street, Johannesburg, on Wednesday 30 March at 8 p.m. The subjects under discussion will be: 'Clinical and experimental hypnosis in contemporary behavioural sciences' by Dr. Le Jeune; 'Dental applications in hypnosis' by Dr. S. Rootenberg; and 'Hypnosis as a physiological state' by Dr. B. W. Levinson. Dr. C. Shubitz will be in the chair.

* * *

Dr. L. J. A. Loewenthal, dermatologist, of Johannesburg, has been elected a Member of the Board of Directors of the recently founded International Society of Tropical Dermatology. He has also been made a Corresponding Member of the Austrian Dermatological Society.

* * *

Society for Clinical and Experimental Hypnosis of South Africa (Southern Section). A meeting of this Society will be held in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, on Tuesday 29 March at 8.15 p.m. Dr. M. Herman will speak on 'Hypnosis—its nature, phenomena and application'. The telephone number of the venue is 5-1111.

* * *

Northern Transvaal Branch (M.A.S.A.). The Branch office is to be moved from Room 4 to Room 15 in the Administrative Building of the General Hospital. Telephone 2-9741, extension 211.

Noord-Transvaalse Tak (*M.V.S.A.*). Die Takkantoor word verskuif vanaf kamer 4 na Kamer 15 in die Administratiewe Gebou van die Algemene Hospitaal. Telefoon 2-9741, uitbreiding 211.

* * *

Bantu Medicine. A Medical Conference on Bantu medicine will be held at Pietersburg, Transvaal, on 5 and 6 April 1960. Information may be obtained from the Hon. Secretary, Dr. N. J. le Roux, at P.O. Box 128, Pietersburg. Among the items scheduled for the Conference is a symposium on 'The influence of tribal habits and beliefs on hospital and medical practice'; case discussions; and lectures by Dr. J. H. S. Gear (Recent advances in the diagnosis and prevention of virus diseases with special reference to Southern Africa), Prof. H. W. Snyman (Hartsiektes by die Bantoe) and Dr. B. A. Dormer (Some non-tuberculous chest diseases in the Bantu).

Various social functions have also been organized, such as a braaivleis and buffet supper.

* * *

Nagraadse Skool Beplanningskomitee, Bloemfontein. Op 30 Januarie 1960 is die volgende twee bydraes in die Algemene Praktisynsreeks in die *Tydskrif* geplaas: ‚Diagnose van die akute buik' deur dr. von W. Eybers, en ‚Gynaecological and obstetrical causes of acute abdominal or pelvic pain' deur dr. J. P. Theron; en op 13 Februarie 1960 is dr. J. D. Meyer se bydrae oor ‚Interne aspekte van die akute buik' geplaas. Ten einde groter duidelikheid te verkry oor die agtergrond waarteen hierdie bydraes gelewer is, dien gemeld te word dat hulle deel uitgemaak het van 'n simposium oor ‚Die akute buik' wat gereël is gedurende die opknappingskursus wat die Nagraadse Skool Beplanningskomitee op 25-27 Junie 1959 te Bloemfontein georganiseer het.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

ELECTROPHORESIS INSTRUMENT 35

VEB Carl Zeiss Jena introduce their new Electrophoresis Instrument 35 and supply the following information:

This small instrument for free electrophoresis corresponds in design and applicability to the Macro-electrophoresis Instrument (*Jena Review*, **3**, 109, 1958).

Two factors played a decisive part in its design, viz.: reduction of the quantity of material required and increase in the working capacity of the instrument by cutting the time of an analysis.

The cuvette of the Electrophoresis Instrument 35 has a capacity of only 1·335 ml. as compared to the 15 ml. of the Macro-electrophoresis Instrument. The liquid examined is therefore reduced from 9 to under 1 ml. The useful height of the cuvette, however, still amounts to 35 mm. as compared to the 84 mm. of the Macro-Electrophoresis Instrument. Due to the lower depth of the cuvette, the separating power lags only slightly behind that of the Macro-Electrophoresis. Separating time is reduced to an average of 40 minutes, the time of dialysis to 1 hour.

For observation and measurement the Philpot-Svensson Schlieren instrument is used. This can be converted into a Rayleigh Interferometer by a simple switching procedure indicated by Svensson. This makes it possible to form an image not only of the gradient curve ($\frac{dn}{dx}$/x diagram) but also interferometrically of the concentration curve (n/x diagram). The gradient curve is used only for the defining of fractions whilst the content of substance of each fraction results from the shift of the interference fringes. This makes the evaluation not only more accurate but also considerably simpler and it takes only a few minutes. With its efficient design and easy operation the Electrophoresis Instrument 35 meets all demands made on a modern laboratory instrument.

Further information may be obtained from Messrs. Optolabor (Pty.) Ltd., P.O. Box 1820, Johannesburg, the South African agents for VEB Carl Zeiss Jena.

BOEKBESPREKINGS : BOOK REVIEWS

RADIOTHERAPY

A Short Text-book of Radiotherapy for Technicians and Students. 2nd edition. By J. Walter, M.A., B.M. (Oxfd), M.R.C.P. (Lond.), F.F.R., D.M.R.E. (Camb.) and H. Miller, M.A., Ph.D. (Camb.), F.Inst.P. Pp. xi + 527. 303 illustrations. 56s. net. London: J. & A. Churchill Ltd. 1959.

Since its first appearance in 1950 this work of collaboration between a radiotherapist and a physicist has deservedly gained the reputation of being the standard elementary text-book of radiotherapy.

The second edition, recently published, has been extensively revised to take account of new concepts in radiobiology, dosimetry, and modern developments in technology, including supervoltage apparatus. There are new sections devoted to hormone therapy, chemotherapy, radio-active isotopes and radiation hazards.

The growing importance of irradiation as an ancillary therapeutic measure in medicine and surgery, the wider use of radioactive isotopes, public concern with the increasing danger of overexposure to internal or external sources, have resulted in a demand for a better understanding of the fundamental biological effects of radiation by workers in all fields of medicine. Moreover, such knowledge would tend to foster a freer interchange of ideas between radiotherapists and their medical and surgical colleagues, thereby promoting the ideal cooperative approach towards the treatment of cancer patients. Drs. Walter and Miller are to be congratulated on adequately fulfilling this need.

The present volume admirably serves the vital function of providing a clear and succinct presentation of radiation physics, radiobiology and radiation therapy. The text which is basic and practical, is devoid of complexities and bewildering mathematical formulae, and should be readily understood by the non-specialist in radiotherapy. The reviewer warmly recommends the publication to student radiographers, hospital physicists, tutors, hospital administrators, consultants, and registrars. D.D.

TEXT-BOOK ON DIABETES

The Clinical Syndrome of Diabetes Mellitus. By John Lister, M.A., M.D., M.R.C.P. (Lond.). Pp. x + 234. 34 illustrations. 35s. net. London: H. K. Lewis & Co. Ltd. 1959.

I think the author is right in saying that a small general text-book on diabetes should fill a definite want. Dr. Lister (who is also the London correspondent to the *New England Journal of Medicine*) has certainly succeeded in providing a pleasant, simple, clear and common-sense book for medical students and general practitioners. Lists of references are quite considerable, and seem well chosen. These may be of assistance also to the more specialized physician. The historical introduction is nicely written.

In a few matters Dr. Lister is not entirely correct, or seems a little behind the times. Thus chronic pancreatitis (p. 29) is not at all uncommon in this country and characteristically produces *severe* (not mild) diabetes. A weight-reducing diet of 1,000 calories (p. 61) is now generally believed to be inadequate restriction for really obese people. There is no evidence that growth hormone in the human is implicated in any way in diabetes (except in acromegaly), or in prediabetic phenomena. The dietary considerations refer to carbohydrate only—it may well be that a restriction of *fat* intake is of greater importance. This at least merits a discussion. Finally, although the importance of urinary infection is rightly stressed, the great danger of catheterization in the diabetic is not; many of us today avoid this as completely as possible.

Nevertheless this book can be heartily recommended and I intend to recommend it to my students. W.P.U.J.

TUBERCULOSIS NUMBER OF THE WHO BULLETIN

Bulletin of the World Health Organization. Vol. 21, No. 1. Pp. 144. Illustrations. 10s. Geneva: World Health Organization. 1959.

This issue of the Bulletin presents papers on two interesting and widely divergent aspects of the control of tuberculosis.

The first paper is an outline of the result of the first 4-year

follow-up of the 1950-52 mass campaign in Denmark, where intensive work on a nation-wide scale has been going on for years. Arising from this survey the authors suggest the delaying of BCG vaccination of infants and children until the last year of school and the application of the tuberculin test each year to all children. They make the further point that persons of any age with suspicious X-ray lesions, and young people with large tuberculin reactions, should be followed-up systematically—the use of a chemoprophylactic agent may, in this group, further the eradication of tuberculosis.

As a complete contrast to the first, the second paper deals with a pilot scheme among the poorer classes in Madras to compare the advantages of sanitorium and home treatment. The authors found that after 12 months the end results showed only small advantages in the sanatorium group in spite of the fact that the patients on home treatment had unfavourable clinical features at the start of treatment. The small number in the home group (96) made for good control, but the point is brought out that it is difficult to maintain efficient control without adequate supervisory staff, cooperation on the part of patients, and monetary help. J.B.P.

PHARMACOLOGY OF THE NERVOUS SYSTEM

The Effect of Pharmacologic Agents on the Nervous System. Proceedings of the Association for Research in Nervous and Mental Diseases. Vol. XXXVII. Edited by F. J. Braceland, M.D. Pp. xi + 488. 124 illustrations. 34 tables. 108s. net. London: Baillière, Tindall and Cox. 1959.

It is sometimes said that psychiatrists are rapidly becoming pharmacists and biochemists have taken up psychotherapy, but the sneer is misapplied, for it has always been tacitly implied in medical practice and teaching that brain-mind relationships are on interactionist levels. This fine volume is packed with interest for physicians and psychiatrists as well as for neurologists and anyone interested in therapeutics. The three great highlights in the pharmacological approach to the understanding and control of abnormal emotional states have been derived from three different disciplines—physiology (the reticular activating system), therapeutics (the phenothiazine derivatives), and experimental psychiatry (the hallucinogenic drugs); these three divergent roads have recently criss-crossed and now seem to be running parallel to each other.

It would be invidious to pick out any one article in a book which is well up to the high standards of the Association for Research in Nervous and Mental Diseases; the subjects covered range from studies of experimental methods to precise descriptions of the metabolic and physiological problems embraced by the closing arms of neurology and psychiatry. Despite the rapidity of advances in the subjects dealt with in this volume there is no doubt that it will remain a source-book for a long time to come. J.M.MacG.

SPEECH AND THE BRAIN

Speech and Brain-mechanisms. By Wilder Penfield and Lamar Roberts. Pp. xiii + 286. Illustrations. $6.00. Princeton: Princeton University Press. 1959.

'Men ought to know that from the brain and the brain alone arise our pleasures, joys, laughter and jests, as well as our sorrows, pains, griefs and tears. Through it, in particular, we think, see, hear, and distinguish the ugly from the beautiful, the bad from the good, the pleasant from the unpleasant . . . . To consciousness the brain is the messenger'. Hippocrates wrote these words 2,000 years ago and since then all manner of men have devoted themselves to a study of the brain of man and of that very special function of the brain, speech, which uniquely distinguishes man from other living creatures. Penfield, of Montreal, is one of the great figures in medicine of this century and his studies of the living brain in man have added greatly to our knowledge and understanding; this new book, written in conjunction with Lamar Roberts, will add considerably to his stature and place us all even more in his debt. Aphasia is a difficulty in the ideational elaboration of speech and this book deals with the history of its study, with the many theories propounded to account for it, and describes the authors' own investigations and the conclusions they draw from them. Briefly these are: The speech mechanism must function as a whole and is not divisible into restricted functional units, though it is a useful clinical convention to talk of motor and sensory aphasia when one or other aspect of speech is predominantly involved. The left hemisphere is usually dominant for speech regardless of the handedness of the individual, with the exclusion of those who have cerebral injuries early in life. The areas of importance for speech are the posterior temporoparietal, Broca's, and the supplementary motor areas. The book is well written and excellently produced, with many figures, and is essential reading for all who are interested in the brain and the mind. S.B.

BIOGRAPHY OF A MEDICAL IDEALIST

Doctor Squibb—the Life and Times of a Rugged Idealist. By Lawrence G. Blochman. Pp. xii + 371. $5.00. New York: Simon and Schuster. 1958.

There can be few medical men who can claim to have kept a full diary for more than 50 years of their lives. Dr. Squibb did this in spite of a life of activity in practice and in campaigning for pure drugs and the establishment of the US pharmacopoeia. He was the first to invent a suitable mask for ether anaesthesia and was the first man to distil pure ether by means of live steam.

From the thousands of pages of his handwritten diary his biographer has set out a story of contemporary American life as seen through the eyes of a man who was Quaker turned Naval Doctor, and who built up what is now a world-famous pharmaceutical corporation.

It makes fascinating reading. A.H.T.

BRIEWERUBRIEK : CORRESPONDENCE

INCIDENCE OF DIABETES MELLITUS IN ONE DISTRICT OF BASUTOLAND

To the Editor: I was more than interested to note the communications of 30 January and 27 February 1960 from Dr. W. M. Politzer[1] and Drs. H. C. Seftel and G. J. Abrams[2] respectively, regarding diabetes mellitus in the Bantu.

For approximately 15 months during 1957-1958 special attention was paid to all diabetic patients attending the Out-patient Department of the Native Urban Hospital at the Langa Township.

This hospital caters for a self-contained Bantu population, largely Xosas, with only a few Basutos, of approximately 20,000 individuals comprising married couples, their children, and also younger men from the Bantu territories on migrant labour whose wives and families are not with them at Langa. By and large, the whole population uses the facilities of the Out-patient Department so that a reasonable reflection of the diabetic incidence in this group is thus given by the number of diabetics collected, viz. 45. These were all confirmed cases in that all had been referred to the Diabetic Clinic attached to the Out-patient Department of the Groote Schuur Hospital. One proved to be a juvenile diabetic.

The percentage of cases of diabetes for this community is thus in the region of 0·22 which is almost identical with the finding of Dr. Politzer in a rural Bantu population.

It is therefore suggested from the Langa findings that not much store should be laid on the place of domicile when one is dealing with the incidence of diabetes in the Bantu. The migrant Bantu does admittedly constitute, at Langa, the bulk of the population; and this fact could, to some extent, explain the closer proximity of the above figures to those found by Dr. Politzer for a rural Bantu group.

S. Sanders
Medical Officer
City Health Department
'Libertas'
Hertzog Boulevard
Roggebaai
Cape Town

8 March 1960

1. Transvaal Society of Pathologists (1960): S. Afr. Med. J., 39, 95.
2. Correspondence (1960): *Ibid.*, 34, 188.